

EMERGENCIES IN
MEDICAL PRACTICE

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EMERGENCIES IN MEDICAL PRACTICE

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WITH 113 ILLUSTRATIONS
8 IN FULL COLOUR

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PREFACE

A MEDICAL emergency is any condition or circumstance of a patient which calls for immediate action other than surgery. Just what conditions might be included in this definition is a matter of opinion and the doctor's list consisting mostly of absolute emergencies will be shorter than the patient's which includes the relative ones.

Our methods for dealing with urgent and non urgent illnesses differ. In the latter it is not very important whether the diagnosis is made to day or to morrow and so there is time to consider every detail. In the urgent case however a diagnosis must be made at once. We must recognise what matters at the moment and there is rarely time to appeal to special diagnostic methods.

When events are moving fast it is the physician who is deeply versed in the natural history of disease who is likely to be of the greatest service to the patient in peril of his life. In the words of Professor J A Ryle, Eyes without the microscope ears without the stethoscope wits without the help of chemistry and radiology not that we should deny ourselves the proper use of these can often carry us a long way. In an emergency they may have to carry us the whole way.

Another important aspect of emergencies which it behoves us to remember is the anxiety of the patient and his relatives. When summoned we should go at once preserving the equanimity which Osler taught us to cultivate yet avoiding the bustle and bounce against which Trotter warned us. Our attitude should be one of alertness in diagnosis safety in treatment and care in what we say.

With these principles in mind I started some years ago to prepare statements on the treatment of the acute and urgent illnesses which came my way. I had found that clear and concise instructions on what to do in these emergencies were scattered amongst the sections on general therapeutics in medical text books.

When I reviewed my notes with a view to publication it was

apparent that I could not hope to deal adequately with all aspects of the subject. I therefore sought the help of colleagues in the various fields of medicine. Our aim has been to provide information as accurate and explicit as possible for the practitioner and hospital physician faced with an acutely ill patient or a critical situation. As a mere list of treatments would be dull, some discussion has been included on diagnosis and the principles on which treatment is based. Details are given of important practical points such as where the rarer drugs may be obtained and the telephone numbers which might be needed in an emergency. References to original work have been included only in a few special instances.

Doses in the text are mostly stated in the Apothecary system. As their *exact* metric equivalent is often inconvenient to measure this is not given and those wishing to employ the Metric system should refer to the conversion table on pages 372 and 373.

Many colleagues in many hospitals have given helpful criticism and advice and to them I am very grateful. In my own sections I have had the advice of—Dr Robert Coope, Dr H. L. Marriott, Mr Willard Maclean, Mr Kenneth Bullock, Ph.D. and Mr D. Harcourt Kitchin and several of my colleagues in the hospitals formerly maintained by the Middlesex County Council, especially Dr A. G. Hounslow, Mr Tiberio Iattu and Dr F. Avery Jones.

Between the contributors themselves there has been much give and take in our efforts to produce a useful book.

It is fitting that I should acknowledge the inspiration derived from my former teachers in Liverpool, Emeritus Professor John Hay and Professor Henry Cohen and I recall with gratitude the stimulus received from my friend Hamilton Bailey, especially in the earlier stages of the work.

Professor Stanley Alstead, late of Inverness, recently appointed to the Regius Chair of Materia Medica and Therapeutics in the University of Glasgow and Dr R. V. Dent, my first assistant, have shared the burden of proof reading. The arduous work of typing and retyping has been willingly done by my records clerk, Mr Anthony Foster. Throughout my editorial labours my wife has given me continuous encouragement and help. I would also like to acknowledge the assistance I have received from Mr G.

F Home Librarian of the Royal Society of Medicine and the kindness and consideration in trying circumstances of Mr Charles Macmillan of Messrs E & S Livingstone

Several of the illustrations have been drawn by my wife and Miss D M Barber and for permission to use other illustrations I am indebted to various publishers

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June 1948

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THE HAZARDS OF MEDICAL PROCEDURES

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OTHER (NON SURGICAL) ABDOMINAL EMERGENCIES

RESPIRATORY EMERGENCIES

FITS FAINTS AND UNCONSCIOUSNESS

MEDICO LEGAL AND OTHER NON CLINICAL EMERGENCIES

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CHAPTER I

The Emergency Bag

EVERY practitioner who is liable to be called in an emergency should have ready for immediate use an "emergency bag" As experience and skill are at least as important as the contents

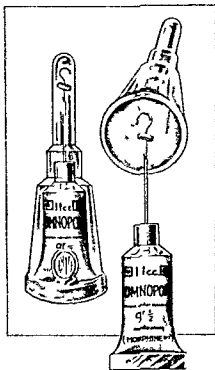


FIG 1
The tubunic ampoule
(*Surgery of Modern Warfare*)

of the bag only the drugs and equipment with whose use he is familiar should be included The following list is suggested —

Drugs

Morphine tablets gr $\frac{1}{4}$

Atropine tablets gr $\frac{1}{100}$ Tubunic ampoules (Roche Products Ltd, Welwyn Garden City) containing these drugs are very convenient (Fig 1)

Nikethamide 2 c cm ampoules

Solution of Adrenaline Hydrochloride B P in bottle or ampoules

Paraldehyde 4 fl oz in coloured glass bottle

Chloroform 6 fl oz

Soluble Hexobarbitone Two 0.5 gm phials and two 5 c cm phials of distilled water

Fifty per cent sterile solution of dextrose 20 fl oz

Pethidine 1 c cm in ampoule (containing 50 mgm)

Insulin One 5 c cm bottle of double strength (40 units per c cm)

Antidiphtheritic serum Six ampoules containing 8 000 units each

Chloral hydrate solution 4 fl oz of a solution containing 30 gr to $\frac{1}{4}$ a fl oz flavoured with syrup of ginger

Universal antidote (page 6)

Sulphamethazine Twenty 0.5 gm tablets

Sulphamezathine sodium Four ampoules of 3 c cm (—1 gm) each

Soluthiazole Four 5 c cm ampoules—each equivalent to 1 gm of sulphathiazole

Penicillin Two vials of 500 000 units each

A bottle of antiseptic such as chlorocresol B P (Dettol)

Appliances

A 2 c cm and a 20 c cm Record syringe fitted with hypodermic and serum needles respectively and stored in spirit proof cases

Sterile water in rubber capped bottle

One teaspoon

Rubber tourniquet

Rubber catheter

Rubber gloves and three thin rubber finger stalls

Cotton wool

Two stomach tubes (adult and child) with funnel

Mouth gag and tongue forceps

Two lumbar puncture needles (Barker Harris) dry sterile in tubes

Sparklet carbon dioxide apparatus

French's venesection needle

Anæsthetic mask

Two throat swabs

Thermometer

Stethoscope

Diagnostic set

Reflex hammer

Two screw-top specimen bottles

Two urgency orders (Shaw & Sons, 7 Fetter Lane, E C 4
Tele CENTral 8171)

The drugs and appliances required for emergencies in hospitals are chiefly those mentioned in the chapter on Practical Procedures (*page 372*) and the appendix on drugs (*page 449*)

J W CHEETHAM

CHAPTER II

Acute Poisoning

WHILE the fact that a patient has been poisoned is generally obvious this is not always so and hence it is important when confronted with an obscure sudden illness that the possibility of poisoning should cross one's mind before valuable time is lost (*For poisoning on board ship see page 395*)

Blood albumin and sugar in the urine and even extensor plantar responses do not necessarily mean that the illness is natural since poisons may be responsible

It is more important to know what effects a poison is producing and how it was taken than to know exactly what the poison is H L Marriott has pointed out that the old antidotal treatment of poisoning was often impracticable because the poison was sometimes unknown or if known its antidote was forgotten or if remembered not at hand The two important principles in treatment are —

- (1) To remove or neutralise the poison
- (2) To keep the patient alive by dealing with the effects of poisoning

REMOVAL OR NEUTRALISATION OF POISONS

Inhaled poisons (usually carbon monoxide)

Carbon monoxide (CO) is poisonous because having 300 times the affinity of oxygen for haemoglobin it produces chemical asphyxia by converting oxyhaemoglobin (HbO_2) to carboxyhaemoglobin (HbCO) CO also interferes with the release of oxygen from HbO_2 Children are affected by CO earlier and more severely than adults

The reaction between CO and Hb is reversible and is more rapid if pure oxygen is breathed To promote recovery it is essential to increase greatly the pulmonary ventilation and this is rapidly achieved by adding 7 per cent of CO_2 to the oxygen

Diagnosis —CO poisoning is often obvious—as when a patient is found in a gas filled room but poisoning may occur from the

CO evolved from a bucket of hot coals or charcoal burning in a restricted supply of oxygen. If there is any doubt, treat for CO poisoning by giving oxygen and CO_2 . These cannot harm the patient, whereas delay in their use might be serious in real CO poisoning. Spectroscopic tests for HbO_2 and HbCO may be done later, on a dilute solution of blood in distilled water, but they need expert interpretation. Both HbO_2 and HbCO show two absorption bands when the solutions are examined spectroscopically, though with low concentrations these may be difficult to detect. If a few drops of ammonium sulphide are added the two bands remain when due to HbCO , but become one band (reduced Hb) if only HbO_2 was present.

Treatment—If a gas filled room has to be entered go in crouching (CO is slightly lighter than air). The rescuer should be 'roped' so that he can be dragged out if he is overcome and does not return in the agreed time—say one minute. Turn off the gas and drag the patient out by the heels. Clear the airway by pulling forward the tongue and using a swab. Beware of injuring the tongue as it may swell rapidly. Do not use the crushing type of tongue forceps.

If breathing is very shallow, apply artificial respiration preferably in the prone position (*Schäfer's Method page 395*) so that secretions can escape. Give seven per cent CO_2 in 93 per cent oxygen by an oro-nasal mask.

Swallowed poisons.

In all cases, except in those in which corrosives—strong mineral acids and strong alkalis—have been swallowed, the stomach should be washed out (*page 391*). This should be done however long the interval since the poison was taken, however much vomiting has occurred and however well the patient seems.

Only when a stomach tube is not available should an emetic be given. Apomorphine hydrochloride gr $\frac{1}{8}$ subcutaneously is the most convenient and least depressing.

Gastric lavage is much more important than the administration of an antidote. At the same time there is something to be said for giving a dessertspoonful of the following Universal Antidote while preparations are being made for a stomach wash out.

Activated charcoal (powdered)	2 parts
Magnesium oxide	1 part
Tannic acid	1 part

Most of this dose will be returned with the stomach washings. When lavage is completed a second dose should be given.

To neutralise corrosive acids give four teaspoonfuls of magnesia in a few ounces of water to form a paste. Soap suds or washing soda may be used instead.

To neutralise corrosive alkalis give 3 fl oz of vinegar or lemon juice for the organic acids which they contain. White of egg may be used in either case to form a relatively inert coagulum.

KEEPING THE PATIENT ALIVE

The life of the patient is immediately endangered by one or more of the following results of poisoning —

- (1) Asphyxia
- (2) Coma
- (3) Dehydration and chloride loss
- (4) Pain and shock
- (5) Delirium and convulsions

Each of these may demand prompt treatment irrespective of the poison responsible.

Asphyxia

This may be chemical (as in CO poisoning) obstructive (as from inhaled vomit inflammatory exudate and œdema) or due to depression of the respiratory centre as in coma. The essentials of treatment are —

- (1) To maintain a good airway
- (2) To apply artificial respiration
- (3) To administer oxygen or if breathing is feeble oxygen with CO

Coma (For other causes see p. 146)

(1) Give strychnine hydrochloride gr $\frac{1}{4}$ in 5 c cm saline by slow intravenous injection and leptazol 2 c cm intravenously repeating the latter every few minutes if necessary.

If these measures fail picrotoxin should be used particularly in the deep coma of barbiturate poisoning. 1 to 2 mgm should be given intravenously every minute until the corneal reflexes

appear or twitchings occur. A total dose of 1 000 mgm may be given in divided doses over a period of 48 hours.

(2) Perform lumbar and cisternal puncture (*pages 374 and 378*) and repeat every eight hours. This removes the poison containing fluid and also relieves the raised intracranial tension which is often present. Fluid should be removed at least until the pressure is normal and in deeply comatose patients until no more flows.

(3) Maintain an airway and perform artificial respiration if necessary. Coma is nearly always accompanied by some degree of asphyxia.

(4) Unless the patient is taking fluid by mouth within eight hours put up an intravenous saline drip.

(5) It is wise to give a sulphonamide at the outset as comatose patients readily develop pneumonia. Should urinary excretion be inadequate or doubtful it would be wiser to use penicillin.

Dehydration and chloride loss

Profuse vomiting and diarrhoea will soon produce a state of collapse from dehydration and chloride loss. The urine output falls. The skin becomes lax and the eyes sunken. Thirst is intense and cramps occur.

It is best to give half strength normal saline (40 grains to one pint or 0.45 gm in 100 c cm) sweetened with glucose and flavoured with orange or lemon. A gallon (4½ litres approximately) may be needed. The drinking of fluid should go on in spite of vomiting. It is nearly always necessary however to give normal saline intravenously.

Pain and shock

These are symptoms of corrosive poisoning and should be treated by giving morphine gr ¼ subcutaneously and fluids intravenously.

Delirium and Convulsions

These are not common symptoms of poisoning but when present they contribute to a fatal result. One of the easiest ways of quietening a violent patient is to inject intramuscularly into whatever part presents 5 c cm of paraldehyde straight from the bottle.

Pernocton—a barbiturate—may be injected intravenously giving 1 c cm per minute until the patient sleeps. Up to 8 c cm

may be needed. Or it may be given intramuscularly in doses of 1 c cm per 20 lb body weight.

The above principles apply to all cases of acute poisoning.

Aspirin, barbiturate and methyl alcohol poisoning are mentioned in more detail because they are more commonly encountered. Poisoning by mercurial salts for which new and efficient treatment is available is described on page 238.

ASPIRIN POISONING

This presents two features—dehydration and acidosis—calling for special treatment.

- (1) Dehydration from sweating (which may be so intense as to soak the mattress completely) must be made up by giving intravenous saline, dextrose or plasma, bearing in mind the danger of a too copious infusion. (*See Circulatory Overloading* page 30.)
- (2) Acidosis indicated by increased depth of respiration is combatted by the use of intravenous sodium bicarbonate and sodium lactate in amounts sufficient to bring the alkali reserve of the blood back to normal.

The amount required is best determined from the carbon dioxide combining power of the plasma. Normally 53 to 77 volumes per cent it may be as low as 30 volumes per cent in aspirin poisoning. Two methods are available.

(1) The alkali reserve of the blood is estimated by the van Slyke or other method and expressed as volumes of CO_2 per cent or as mille mols per litre ($\frac{\text{mM}}{\text{L}}$). The patient's weight is next obtained or estimated. The desired rise of plasma CO_2 required is decided and by using a nomogram (Fig. 2) the amount of isotonic ($\frac{\text{m}}{6}$)

bicarbonate or lactate necessary to produce it is read off.

(2) By calculation. Aspirin (acetylsalicylic acid Mol. wt. 180.1) yields on hydrolysis in the body equimolecular amounts of salicylic acid and acetic acid. The molecular weight of sodium bicarbonate is 84 and of sodium lactate 112. Hence every 180.1 grams of aspirin absorbed will need for neutralisation 84 grams of sodium bicarbonate with 112 grams of sodium lactate or double these amounts of either.

In the body two complicating factors upset this simple calculation. (a) The normal plasma bicarbonate already present. (b) the fact that the acetic acid released forms sodium acetate which is itself oxidised to bicarbonate. These may be neglected in the calculation. Example—If a patient swallows and absorbs one hundred 5 grain aspirin tablets—say 30 gm—the amount of bicarbonate required to neutralise the acid formed would be 2 molecules of bicarbonate for each molecule of aspirin. Hence 30 gm aspirin would require 28 gm of sodium bicarbonate or 37 gm of sodium lactate. In practice it is wise to use both the quickly acting bicarbonate and the more slowly acting lactate (i.e. to give 14 gm sodium bicarbonate and 18.5 gm sodium lactate).

Failing any of these aids the reaction of the urine should be our guide. Samples obtained by means of a self retaining catheter should be tested every 15 minutes. 20 gm of

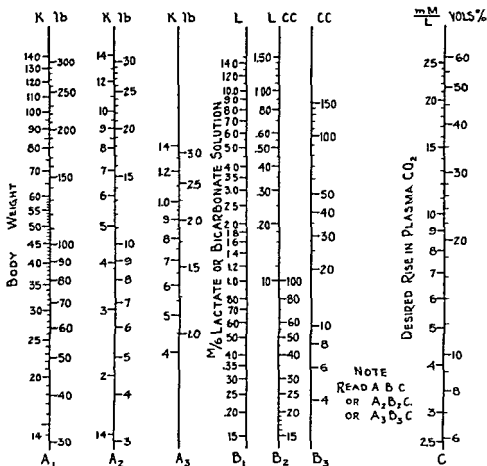


FIG 2

Nomogram showing amount of m/6 lactate needed to produce a desired rise in plasma CO_2 .

sodium bicarbonate in about 400 c cm of water should be given intravenously by a syringe and then an intravenous drip of $\frac{1}{2}$ normal saline with 5 per cent dextrose and 20 gm of sodium bicarbonate continued. For the next infusion one quarter strength normal plasma may be used.

Intramuscular injection of morphine gr $\frac{1}{4}$ to $\frac{1}{2}$ or paraldehyde 5 c cm may be necessary to render the patient quiet.

enough for intravenous therapy Warmth oxygen and other measures as described under Medical Shock (page 109) may be used as indicated

Two further urgent symptoms might arise in aspirin poisoning A violent paroxysm of asthma might be caused in an aspirin sensitive asthmatic Hæmatemesis is also a possibility because aspirin is a gastric irritant and also decreases the coagulability of the blood

BARBITURATE POISONING

Poisoning by barbiturates presents as profound coma with flaccid limbs The plantar reflexes may be extensor

(1) Repeated gastric lavage is important but plain water only should be used Bicarbonate should be avoided as it increases the solubility of the barbiturate Sweetened coffee about one pint and white mixture B P C 2 fl oz should be left in the stomach It must not be forgotten however that it may be dangerous to leave much fluid in the stomach of a comatose patient lest it be regurgitated and inhaled Careful watching is therefore necessary

(2) Colonic lavage should be used

(3) Lumbar and cisternal puncture should be performed every twelve hours and as much C S F as possible drained

(4) Analeptic injections Give leptazol 2 c cm intravenously and strychnine hydrochloride gr $\frac{1}{4}$ intravenously and repeat hourly if necessary If these fail to lessen the depth of coma give picrotoxin (see *Index* page 6)

(5) An adequate airway must be maintained This may entail the passage of a nasal endo tracheal tube and keeping it clear by suction through a catheter

(6) If coma persists after 24 hours start giving a sulphonamide or penicillin

METHYL ALCOHOL POISONING

This is usually the result of drinking methylated spirit Its seriousness depends on the fact that the toxic methyl alcohol is only slowly and partially oxidised in the body to formic acid and formates which are also toxic

Symptoms may be immediate or delayed They include headache vertigo vomiting and coma Dyspnoea from acidosis

is marked. Dimness of vision progressing to blindness due to an acute papillitis is not uncommon.

Treatment—In addition to the general measures for the treatment of poisoning, the severe acidosis must also be combatted as described under aspirin poisoning.

Whiskey in doses of 1 fl. oz. every four hours is recommended since its ethyl alcohol is thought to displace methyl alcohol from the tissues. The eyes should be covered until all danger of retinitis is passed.

C. ALLAN BIRCH

CHAPTER III

The Hazards of Medical Procedures

ACCIDENTS WITH NEEDLES

Broken needles

SHORT needles usually break at the junction of the shaft and the butt. Long needles may rust internally and break anywhere. Nickel needles bend rather than break and should be used for lumbar puncture if sudden jerks are expected. All needles should be tested before use and a needle should never be inserted as far as it will go. A guard may be placed on the needle to prevent too deep insertion. Should a needle break in the tissues and sufficient project it may be removed by forceps but more often no part remains visible though pressure on each side of the puncture may reveal the end. Beware of making a small incision and poking about. Prevent the patient from moving the part tell him what has happened but do not admit negligence or offer compensation. Have the needle removed at a planned operation. If the patient is too ill for this say nothing and leave him alone. He may die. If he recovers from his illness always have him X rayed before exploring. Small portions of the needle have been left in the body and even in the pleural cavity without causing untoward effects.

Paravenous injections

Many substances commonly given intravenously such as neoarsphenamine some of the soluble sulphonamides and iodoxyl (Uroselectan) are highly irritant if they leak into the subcutaneous tissues. Diodone a compound allied to iodoxyl has the advantage of being non irritant and may be given subcutaneously. Suspected leakage of iodoxyl should be confirmed by radiography (Fig. 3). Absence of X ray evidence of leakage may be useful to the doctor in a subsequent legal action. A glass adaptor between the syringe and the needle makes it easy to see the blood on withdrawing the piston and thus indicates that the vein has been entered. This is otherwise impossible when using dark coloured solutions.

Treatment consists of immobilising the limb and applying warmth. Some of the solution may be aspirated from the subcutaneous tissues.



FIG. 3

Showing subcutaneous leakage of iodoxyl at the elbow
(Dr P H Walter)

Entry of an artery instead of a vein

This may happen when an aberrant artery lies between the fascia and the skin when a cubital vein is transfixed or when exploring for a vein in a plump ante cubital fossa. Intense burning pain is felt in the hand and thrombosis and gangrene may occur later.

Two simple precautions indicate that the vessel in question is an artery and not a vein. Careful palpation usually reveals pulsation of an artery and after puncture the blood withdrawn

is bright red. It is always wise to wait a few seconds after commencing a supposed intravenous injection and proceeding only if no untoward effects appear.

Pericardial hæmorrhage.

This is one of the remote risks of intracardial injection of drugs such as adrenaline (for cardiac arrest) or 2 per cent

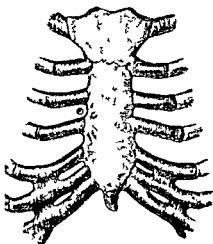


FIG. 4
Showing entry point for injection of
right auricle
(Hamilton: *Bull. N.-Emergency* 5: 171)

procaine (for ventricular fibrillation). It may also occur but is less likely when tapping a pericardial effusion by the anterior route. Pressure of blood in the pericardial sac rather than its total amount is responsible for cardiac tamponade, and as little as 200 c.c.m. has caused death.

Hæmopericardium from puncture is less likely to occur when the auricles rather than the ventricles are injured. As the auricles are also more sensitive to mechanical stimuli than the ventricles, needling and injection are more effective and less dangerous if an auricle is entered. The right auricle should be chosen. A long needle such as a lumbar puncture needle is inserted in the third right interspace at the upper border of the fourth rib close to the sternal margin and directed backwards and slightly inwards (Fig. 4).

Lumbar puncture (For technique see page 374)

Too much preliminary prodding of the back may precipitate hysterical phenomena in susceptible patients as may also the painful periosteal injury of clumsy manipulation

Permanent paralysis has resulted in a few cases It may have been caused by intrathecal bleeding though this is usually harmless or causes only transient root irritation A more probable cause is progression of a pre-existing lesion Hence it is important to record carefully the physical signs present before puncture

If lumbar puncture is performed on a patient with raised intracranial pressure from a subtentorial tumour herniation of the medulla into the foramen magnum (foraminal crowding) may result Similarly in cases of supratentorial tumour herniation of the uncus of the hippocampal gyrus into the tentorial hiatus may occur Withdrawal of minimal volumes of C S F provides no guarantee that these serious complications will not supervene as leakage through the meningeal puncture may continue for some hours

Warning signals are an abrupt cessation of fluid flow followed by neck pain and dysphagia Respiratory failure and death follow in minutes or hours Similar sudden collapse without lumbar puncture may occur in patients with raised intracranial pressure and emphasises the precarious position of these patients

Prevention of these accidents lies in removing as little fluid as possible A Dattner (double) needle is specially useful in these doubtful cases (page 378)

When collapse follows lumbar puncture withdraw the needle elevate the foot of the bed and give nikethamide 2 c cm intravenously Arrange for a neurosurgeon to perform ventricular tapping and while waiting for this give 500 c cm of 50 per centucose intravenously

Cisternal puncture (For technique see page 378)

An emergency may follow cisternal puncture if the needle is inserted more than 2 cm after the occipitoatlantal ligament is pierced In this case the medulla is injured—usually with fatal results Deviation of more than 18° from the mid line may result in damage to a vertebral artery with resulting subarachnoid haemorrhage This may also be due to damage to abnormal

vessels inside the foramen magnum. As these are not uncommon, cisternal puncture is best avoided unless clearly indicated. In either of these emergencies little can be done beyond the giving of analeptics.

EMERGENCIES FOLLOWING LOCAL ANÆSTHESIA

Collapse, convulsions, coma and death may follow the use of local anæsthetics, particularly cocaine, which should generally be avoided in favour of procaine (for injection) and amethocaine (for surface application). Minor early manifestations are pallor, nausea, weakness, restlessness, facial twitching, sweating, tachycardia and dilated pupils. Untoward effects arise from —

- (1) Increased individual susceptibility or actual idiosyncrasy.
- (2) Faulty labelling of solutions so that cocaine is used in error.
- (3) Excessive dosage. The limits are variable but the total quantity of cocaine hydrochloride used should not exceed 0.06 to 0.1 gm (1 to 1½ grains). The maximum safe dose of procaine hydrochloride by injection is 1 gram but it is wise to use less. Fifty c cm of a 1 per cent solution or 100 c cm of a 0.5 per cent solution are usually sufficient. These figures refer to adults; children are less tolerant.
- (4) Too rapid absorption. Cocaine is rapidly absorbed and should never be given by injection. It should be reserved for use in the nose and throat and the eyes and avoided in places covered by other than stratified epithelium. Urethral injections are specially dangerous and more so if a stricture is present or trauma has occurred.

Local anæsthesia of the larynx may be followed by intense spasm of the cords. (See *Bronchoscopy* page 25.) Patients with myasthenia gravis are specially liable to collapse after injection of procaine or cocaine.

Treatment — If cocaine is used, it is wise to give a quickly acting barbiturate such as amytal gr ½ to 3 an hour before hand, since this diminishes the risk of convulsions. Should they occur, they can be immediately cut short by the slow intravenous injection of 5 per cent thiopentone (pentothal). 3 to 5 c cm are usually enough. Collapse and coma, however, call for the use of —

- (1) Artificial respiration, preferably by the rocking method (page 399).

- (2) Seven per cent CO in oxygen administered by a mask (page 411)
- (3) Injection of leptazol 2 c cm intravenously or intramuscularly and repeated if necessary. Failing this strychnine gr $\frac{1}{8}$ may be given intravenously.

If death from respiratory depression seems imminent the injection into the cisterna magna of 1 grain of ephedrine hydrochloride in a few c cm of normal saline has been recommended.

EMERGENCIES ARISING DURING PNEUMOTHORAX AND PNEUMOPERITONEUM THERAPY

Pleural shock

In rare cases collapse and even death have followed immediately on puncture of the pleura. This has been attributed to pleural shock but in many cases so diagnosed the symptoms have clearly been those of cerebral air embolism. The existence of shock apart from embolism has been seriously disputed but in the following case brought to my notice by Dr A G Hounslow true pleural shock seems to have been responsible.

A woman aged 25 suffering from pulmonary tuberculosis had the left phrenic nerve crushed uneventfully under local anaesthesia with 1 per cent procaine. About two months later it was decided to induce a left artificial pneumothorax. Procaine 1 per cent was injected into the skin and subcutaneous tissues down to the pleura using 1.5 c cm in all. Within half a minute of withdrawing the needle—a fine hypodermic—and before any other needle was inserted she looked up and said 'Is it all over?' and then collapsed. There were no convulsions. She was pale and the pulse was imperceptible. Heart sounds could not be heard. In spite of nikethamide subcutaneous and intracardial adrenaline and artificial respiration (20 minutes) fragmentation of retinal vessels was seen and treatment was abandoned.

At autopsy nothing abnormal was discovered but unfortunately the technique for detecting cerebral air embolism was not used.

Treatment—Nothing useful can be done in such a case but in the less severe cases which resemble vaso-vagal faints smelling salts, brandy and leptazol may be used. The head should be placed low. Mild cases showing transient faintness are not uncommon.

Hæmoptysis

This means that the lung has been punctured as shown by an equal manometric swing on each side of zero. Usually nothing more than reassurance is required.

Mediastinal and subcutaneous emphysema

During artificial pneumothorax therapy the needle may damage a pulmonary alveolus and cause interstitial emphysema of the lung. The air may reach the mediastinum and rupture into the pleura causing a tension pneumothorax (*page 103*). The reverse of this phenomenon (pneumothorax rupturing into the mediastinum) does not occur.

Subcutaneous emphysema localised to the site of the puncture does not cause urgent symptoms. When present in the neck it means that mediastinal emphysema is also present (*page 103*). Urgent symptoms are rare because escape of air into the neck relieves the mediastinal pressure. Air may spread to the retroperitoneal tissues and cause abdominal pain. No treatment beyond the use of analgesics is required.

Pneumoperitoneum

Rarely this may be induced inadvertently if the pneumothorax needle is inserted too low. Failure to obtain a satisfactory manometric swing and the disappearance of liver dullness should indicate what has happened. There may be pain in one or both shoulders but if the phrenic nerve has been previously crushed pain will be confined to the opposite side. No special measures are indicated.

When inducing therapeutic pneumoperitoneum the bowel has on a rare occasion been injured as shown by the presence of faecal matter in the needle. No harm results and no treatment is indicated but a period of observation is advisable. Cardiac air embolism has occurred from liver injury. This can be more readily avoided by inserting the needle under the *left* costal margin.

Occasionally air has passed from the peritoneal cavity presumably through the oesophageal hiatus and reached the mediastinum. When pressure is not released by an escape of air into the neck or the retroperitoneal tissues acute symptoms may arise (*for treatment see page 103*).

AIR EMBOLISM

Air embolism is of two clinical types—cardiac and cerebral.

Cardiac air embolism

This accident has resulted from numerous procedures such as

operations on the neck and intracranial venous sinuses, irrigation of the maxillary antra and a submaxillary abscess, Eustachian tube insufflation filling the bladder with air, æro urethroscopy in the presence of urethral hæmorrhage intra uterine injections criminal abortions, vaginal insufflation, tubal patency tests manual separation of the placenta Cæsarean section, and even normal delivery at term

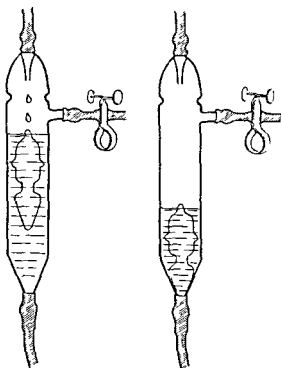


FIG 5

Safety drip chamber to prevent air embolism
(*Lancet Dr W Mustin*)

It may complicate intravenous infusion —

- (1) If the tubing is loose or cracked, or incompletely emptied of air
- (2) If the level of fluid in the bottle falls below the orifice of the exit tube
- (3) If positive pressure is applied when the filter is partly blocked

It is less likely to happen if the clip on the tubing is near the vein rather than near the container, and it may be avoided by using a drip chamber containing a glass float (Fig 5)

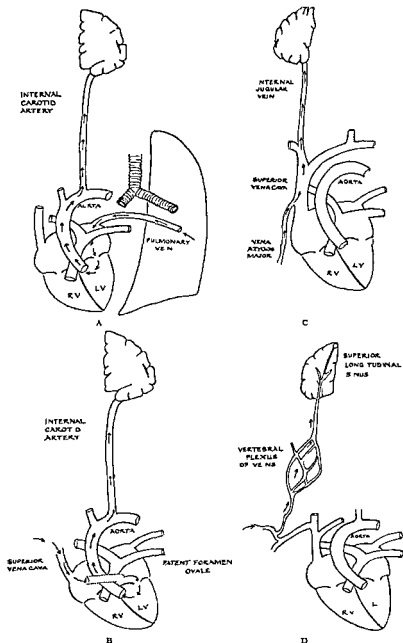


FIG 6

Showing possible routes by which an air embolus may reach the brain

[Continued at foot of opposite page]

Arrangement of the tubing to form a loop below the level of the patient's vein is an additional precaution

It is most likely to be fatal if a large quantity of air rapidly enters a large vein near the heart held open by retraction of the edges of the wound. This mechanism is responsible for the very rapid death which sometimes follows a suicidal throat wound.

A small amount of air injected at the elbow is known to be harmless and it is thought that not less than 480 c cm are needed to produce a fatal result. The rate of injection is important. Large amounts of air have failed to kill animals if injected very slowly.

In all serious cases the right heart is distended with frothy blood. There is usually sudden failure of the pulses and dilatation of the pupils. Auscultation over the heart reveals a characteristic water-wheel splashing sound. The patient takes a few deep breaths and is dead within a minute from acute right-sided heart failure.

The only treatment likely to be beneficial is to aspirate the right heart with a needle and syringe. The needle is introduced between the fifth and sixth cartilages close to the right side of the sternum and when the ventricle is entered the froth is aspirated. The patient should lie on the left side since in this position the bubble forming the air trap is less likely to be in the outflow tract from the right ventricle.

Cerebral air embolism

The symptoms produced are syncope, paralysis and paræsthesiæ and follow involvement of the brain vessels. Embolism

FIG 6—Contd

- (A) Air enters a pulmonary venule when a needle punctures the lung and passes via the left auricle and the left ventricle to the aorta.
- (B) Crossed or paradoxical air embolism. Air enters a systemic venule in the chest wall or elsewhere and passes to the right auricle and via the patent foramen ovale to the left auricle, left ventricle and the aorta.
- (C) Air enters a systemic venule and passes via the azygos vein to the superior vena cava where it rises against the blood stream (especially when the patient sits up) and passes up the internal jugular vein to the brain.
- (D) Air enters a systemic venule and passes via anastomoses with the vertebral venous plexus to the superior longitudinal sinus and so by passes the heart.

of retinal and skin arteries cause visual defects and areas of blanching

The most obvious route is for air to enter a pulmonary vein and go via the left heart to the systemic arteries [Fig 6 (A)] The accident is most often a sequel to the induction of artificial pneumothorax but may complicate the opening of a lung abscess and irrigation of an empyema It does not complicate hæmoptysis When it occurs during pneumothorax therapy it is caused by the needle entering an adhesion containing a radicle of the pulmonary vein On inspiration the pressure in these veins is less than atmospheric and so air is drawn into them Death can therefore follow the injection of a small quantity of air since additional air reaches the vein from the lung itself as well as from the needle In cases where cerebral air embolism has followed puncture of a systemic vein paradoxical embolism via a patent foramen ovale has been postulated [Fig 6 (B)]

There are two other possible routes both venous by which air may reach the brain Air may enter a tributary of the azygos vein or superior vena cava and then rise in the jugular vein against the blood stream to the cerebral sinuses [Fig 6 (C)] This route may explain those cases of cerebral air embolism in which symptoms do not follow quickly on the insertion of the needle but come on later particularly when the patient sits up

A further route is via anastomoses between systemic (or portal) venules and the vertebral plexus of veins [Fig 6 (D)] Air is likely to take this route if for some reason such as coughing or the presence of high intra abdominal pressure as from pneumoperitoneum the blood flow in the inferior vena cava is partially obstructed

Treatment in all these cases consists in immediate cessation of the operation and lowering the head as soon as the patient complains of faintness Even though he quickly recovers it is unwise to resume the operation Morphine should not be given lest depression of the respiratory centre be aggravated

MISCELLANEOUS MEDICAL ACCIDENTS

Chest aspiration

If the pump of a Potain's aspirator is wrongly connected it fills the bottle with air under pressure instead of evacuating it The air may then be introduced into the chest with immediate

collapse of the patient. Treatment consists in reversing the pump and removal of the air quickly. Accidents of this type could be avoided if apparatus were made so that it could not be wrongly connected.

Fractured ribs

Over vigorous artificial respiration in a frail patient may cause fractured ribs. I have known coughing crack a normal rib though this mishap is more likely if a metastasis is present in it.

Bronchography

Acute iodism occurs rarely following injection of iodised oil into the bronchial tree. A test for iodine sensitivity may help to avoid it and will at any rate show that reasonable care was taken. Weak solution of iodine B.P. m 5 in a little milk should be taken on the previous day and the patient watched for a rash or coryza.

Iodism if it occurs is not dangerous though it may cause alarm. Treatment depends on facilitating excretion of the iodide in the urine. Simply stopping administration of the drug and insisting on the patient's drinking freely—about eight pints of fluid a day—suffices for the treatment of most cases. Calamine lotion should be applied to skin eruptions. bullæ may have to be snipped with sterile scissors.

If the cricothyroid route for bronchography is used it is possible to injure the laryngeal mucosa and for the iodised oil to set up œdema. Tracheotomy may be required.

If a laryngeal catheter is used and the bronchi have not been previously emptied by tipping the patient the retained sputum plus iodised oil may be enough to cause respiratory obstruction necessitating abandonment of the operation.

Paracentesis abdominis

If a Southey's silver tube is used and the guard omitted it is possible for the tube to slip into the abdomen at the time of insertion or to part from the tubing and slip in later. It is always wise to search the bed clothes first and then to X ray the abdomen before asking a surgeon to perform a laparotomy.

Nasal feeding

In a feeble infant or deeply unconscious patient it is possible for the tube to enter the larynx. Septic broncho pneumonia may result. Even drowning has occurred.

Before giving a nasal feed the position of the tube should be checked. The mouth and nostrils should be gently closed and the proximal end of the tube held under water or close to the doctor's conjunctiva. If the distal end of the tube is in the trachea a current of air will be detected.

Gastric lavage

This may be very hazardous particularly in a comatose patient. The danger is that upper œsophageal spasm allows the tube to enter the larynx or that in the absence of the cough reflex fluid is regurgitated and enters the lungs. Gastric lavage should therefore be performed with the patient prone and with his head over the edge of the table (*page 391*). Alternatively if an operating table is available the Trendelenburg position may be used. It is a useful safeguard to prove that the tube is in the stomach by showing that fluid which may escape or be aspirated from it after being inserted about 18 inches turns blue litmus paper red.

Electric convulsion therapy

Temporary slowing of the heart and cyanosis are commonly encountered after electrically induced convulsions. Occasionally the heart may stop and an intracardial injection of adrenaline may be needed (*page 14*). Artificial respiration may be necessary to restart breathing after convulsions. Surgical complications namely fractures may also be present but can be avoided by preliminary use of curare.

Gastroscopy

There is slight but definite risk of tearing the œsophagus during gastroscopy especially in elderly kyphotic vitamin starved patients. The risk is increased by the difficulty of passage when the mouth is small and the teeth numerous. With the modern flexible instrument the tear occurs in the region of the post cricoid sphincter where the outer longitudinal muscle fibres of the œsophagus are deficient and the tip of the instrument is apt to be held up by poor swallowing or spasm. Extension of the head at the outset or later in an attempt to increase the field of view adds to the risk.

The symptoms of œsophageal rupture are dysphagia and substernal pain followed by surgical emphysema of the neck. When emphysema is absent (an X-ray examination may be

necessary to exclude it) the other symptoms may be due to oesophagitis. In either case the treatment is to give —

- (1) Penicillin lozenges (500 units in each) one every half hour as far as possible by night and day
- (2) Penicillin 20 000 units three hourly by intramuscular injection or larger doses at longer intervals
- (3) A soluble sulphonamide such as sulphamezathine—soluble up to 12 grams in 24 hours by intravenous injection or soluble thiazole intramuscularly in equivalent amount (*page 441*)

The mouth should be moistened but swallowing of fluids avoided for 24 hours and the fluid requirements made up by rectal or intravenous drip

Bronchoscopy

The hazards of bronchoscopy include —

- (1) THOSE DUE TO THE ANÆSTHETIC —Local anæsthetics particularly cocaine produce their own emergencies (*see page 16*). The use of ether carries the potential risk of explosion. Thiopentone (pentothal) if employed as the sole anæsthetic agent is especially dangerous because in these circumstances any stimulus to the larynx may set up intense spasm. This is a very serious emergency since it quickly leads to asphyxia and prevents the passage of the bronchoscope through which oxygen might be given.

Artificial respiration is of course useless. Relaxation of the cords may occur even when death seems imminent but it should not be awaited since it can be produced by the prompt intravenous injection of 5 mgm of d-tubocurarine chloride. When relaxation occurs the bronchoscope should be slipped through and oxygen blown in at 8 litres a minute. Artificial respiration might also be used at this stage to make recovery doubly sure. Failing all else tracheotomy should be performed and will give instant relief.

- (2) TRAUMA —Damage to the teeth and hyoid bone and perforation of the wall of the respiratory tract are all possible but unlikely. Passage of the bronchoscope through a tuberculous larynx may set up acute oedema and for this reason bronchoscopy is best avoided in patients suffering from laryngeal tuberculosis. If it must be done a tracheotomy set should also be at hand.

(3) **BLEEDING** —Biopsy particularly in the case of an adenoma may lead to profuse bleeding necessitating the aspiration of blood through the bronchoscope. In one case of multiple bronchial papillomata bleeding was stopped by inducing artificial pneumothorax. Coagulants (page 94) should be given. A blood transfusion may be needed.

Retained blood may lead to massive collapse. To prevent blood from passing into the lung drainage should be promoted by placing the patient with his head down. The return to consciousness can be hastened by giving leptazol 2 c cm intramuscularly and the cough reflex is made more sensitive by injecting strychnine hydrochloride gr $\frac{1}{8}$ intravenously.

EMERGENCIES FOLLOWING INJECTIONS

Allergic reactions

SKIN TESTING —Intradermal injection of skin testing material is occasionally followed by alarming reactions characterized by severe local swelling, generalised urticaria and asthma. It is recommended that this procedure should be avoided in children.

A tourniquet should be applied to the limb and a subcutaneous injection of adrenaline given elsewhere. Indeed it is wise to place a sphygmomanometer cuff in position proximal to the site of injection before starting.

TRIFLUUETI INJECTIONS & ALLERGENS —An overdose of allergen or a therapeutic dose in a very sensitive person may cause similar reactions to those described above and should be treated similarly. Non protein substances may be responsible. Inadvertent injection of material into a vein will cause an immediate constitutional reaction but no local swelling.

Treatment —The remedy is adrenaline. A subcutaneous or intramuscular injection of 0.5 c cm of solution of adrenaline hydrochloride (1 in 1000) should be given and repeated if necessary after 15 minutes. The site of injection should be massaged to ensure rapid absorption. In severe and collapsed cases it is sometimes justifiable to give adrenaline intravenously but it is wise to withdraw blood into the syringe first and allow it to mix with the adrenaline before injecting it very slowly. A few drops of sodium citrate solution (3.8 per cent) should first be drawn into the syringe to prevent clotting. If the patient

has recently had a sympathetico mimetic drug such as ephedrine adrenaline should be used with especial caution. Should the response to adrenaline be poor aminophylline 0.2 to 0.5 gram may be given intravenously.

ANAPHYLACTIC SHOCK—This is shown by sudden dyspnoea and pallor coming on during the intravenous injection or shortly after the intramuscular injection of serum in a patient who has had serum of similar origin more than 10 days previously. It is a rare but alarming accident. Marked bronchiolar spasm occurs as shown by diffuse sibilant rhonchi. A similar reaction may follow the *first* injection of a foreign protein especially in people who give a personal or family history of sensitiveness to allergens. Inquiry should always be made for a history of urticaria and asthma.

Treatment—Stop the injection and give adrenaline 0.5 c.c.m. and nikethamide 2 c.c.m. intramuscularly.

The accident can and should be avoided by preliminary testing for sensitiveness. This is most simply done by puncturing the skin of the forearm with a hypodermic needle (this puncture acts as the control) and repeating the puncture a few inches away through a drop of serum. Sensitiveness is shown by redness and weal formation at the site of the second puncture within fifteen minutes. If this is present or if it is necessary to give serum to a patient who has already received a dose more than a week previously the first dose should be 0.1 c.c.m. and this should be doubled at half hourly intervals for about six doses when the remainder may be injected. Each dose should be combined with 0.3 c.c.m. of solution of adrenaline hydrochloride.

Other reactions following injections

Numerous examples could be found of emergencies due to injection of the wrong drug. The label should always be read. If it is missing or illegible the drug should not be used.

Emergencies may also arise from the injection of the right drug by the wrong route. Adrenaline unless very dilute and given very slowly may cause death from ventricular fibrillation if injected intravenously. Less serious results are severe blanching headache palpitation vertigo and occasionally hemiplegia from vascular spasm. As adrenaline is rapidly oxidised

in the tissues useful therapeutic intervention is impracticable. Carbachol similarly given may cause collapse. The clinical picture of fat embolism (*page 101*) may result if oily solutions are injected into veins. Too rapid intravenous injection of many solutions especially cold ones will cause rigors. Ampoules of cold solutions should be warmed in hot water (110° F) before use or the syringe containing the drug should be sealed with a hypodermic needle and placed in hot water. Inadvertent injection of a vaccine into a small vein may quickly produce an alarming result. Material contaminated with bacteria or their products will cause sharp febrile reactions if given intravenously. If symptoms do not abate in a few hours blood should be taken for culture and penicillin therapy started.

Soluble sulphonamides cause disastrous results if used intrathecally and should never be given by this route. Penicillin in strengths greater than 2 000 units per c cm should also be avoided for intrathecal use and the white (crystalline) variety should be used. If however ordinary (yellow) penicillin must be given intrathecally in strong solutions it is best to mix it with cerebrospinal fluid in a syringe before injecting it *very slowly*.

BLOOD TRANSFUSION REACTIONS

These may be immediate or delayed —

IMMEDIATE REACTIONS

When something goes wrong very soon after a blood transfusion is started there are three main groups of conditions which may be responsible

- (1) Non specific factors connected with the apparatus and fluids used
- (2) True blood transfusion reactions due to intravascular hæmolysis
- (3) Circulatory overloading

In addition the risk of transmitting disease may constitute an emergency

Non specific reactions

These include pyrexia, chills and rigors. They result from too rapid transfusion or the presence of foreign protein often of bacterial origin in the transfused fluid. Hæmolysed blood

and blood which has been allowed to warm up may also be responsible. Intense pain in the back, which is so characteristic of intravascular hæmolysis, does not occur.

Treatment—In the absence of evidence of a true blood transfusion reaction it is usually only necessary to slow down the rate of transfusion. If this is ineffective, the source of the blood and of the anti coagulants added should be scrutinised, and if they are suspect a new bottle of blood substituted.

True transfusion reactions.

These are due to faulty grouping and appear usually before 100 c cm of blood have been given. The patient complains of very intense pain in the back and tightness in the chest. He has a rigor, becomes cold, clammy and cyanosed, and may soon die.

In order to avoid such accidents it is important to take special precautions against faulty grouping since this may not always be obvious. One source of error is the fact that group "A" is divided into sub groups " A_1 " and " A_2 " and that there are corresponding sub groups " A_1B " and " A_2B ". " A_1 " and especially " A_2B " cells are relatively insensitive to agglutinins. Hence groups " A_2 " and " A_2B " may be called group "O" and group "B" respectively.

Another example of the need for care is illustrated by the following case. A group "A" patient received four pints of group "O" blood with no untoward results. Next day some group "A" blood was obtained and was found suitable when cross matched with the serum obtained on admission from the recipient, i.e., before any blood had been transfused. Because of the transfusion, almost half of the patient's blood was group "O," i.e., it contained anti "A" and anti "B" agglutinins and so, in effect, a group "O" patient was being transfused with group "A" blood.

Treatment—The transfusion should be stopped, warmth applied, and nikethamide given. If the patient survives the immediate reaction he may soon be found to be in a state of renal failure. This is commonly attributed to blockage of the renal tubules by acid hæmatin. But the severe lumbar pain, evidently of renal origin, occurs before such blockage could arise and this suggests that another mechanism such as renal arterial spasm may be responsible.

Whichever mechanism is responsible rapid alkalisation of the urine and diuresis should be aimed at. Alkali will help to relieve arterial spasm and will hinder the deposition of acid hæmatin. 10 c cm each of isotonic sodium lactate (the theoretical concentration is 1.87 per cent but as the B.P. standard of purity for lactic acid is only 87.5 per cent it is approximate only) and a saturated solution of sodium bicarbonate should be injected intravenously. Alkalinity of the urine is then maintained by giving potassium citrate and sodium bicarbonate gr. 30 of each in an ounce of water every four hours or Citralka (Parke Davis & Co.) half an ounce four hourly. An intravenous saline infusion should be started and a catheter be passed six hourly.

A small transfusion even a 20 c cm syringe of compatible blood promptly given is said to relieve the symptoms but I have no experience of this.

Circulatory overloading

In a previously healthy patient rendered rapidly anæmic and shocked by blood loss the danger of circulatory overloading is slight. Indeed rapid transfusion is desirable to restore the blood pressure to normal. In the chronically anæmic patient with feeble heart muscle the danger of a too rapid transfusion of blood is very real.

Starling's work showed that a rise in venous pressure is associated with increased cardiac output within certain limits the healthy heart adjusts itself to deal with as much blood as may be poured into the right auricle. A stage may be reached however at which this physiological mechanism breaks down it occurs much earlier in patients suffering from anæmia than in healthy subjects and is apt to supervene with dramatic suddenness during a too rapid transfusion of a patient suffering from long standing anæmia. Clinical signs produced are dyspnoea and moist sounds in the lungs. They are liable to appear if the rate of transfusion exceeds 1 c cm per lb per hour (or 0.5 c cm per lb per hour if the hæmoglobin level is 25 per cent or less).

Treatment of circulatory overloading consists in —

- (1) Stopping the transfusion
- (2) Venesection. Remove half to one pint of blood according to the severity of the symptoms and the volume of blood transfused.

- (3) Administration of oxygen
- (4) Mersalyl, 2 c cm intravenously

Transmitted disease.

The risk of transmitting disease from the donor to the recipient is slight if healthy donors only are used, and those who have ever suffered from malaria or who have positive Wassermann or Kahn reactions are rejected. In any case, the resulting situation could hardly be classed as an emergency. The risk of inducing a virulent septicæmia from contamination of stored blood exists, and immediate symptoms would result calling for prompt treatment by penicillin and/or a sulphonamide.

If a recipient is suffering from septicæmia this might be transmitted to the donor if an arm to arm transfusion by the Jubé method were being performed, since it is possible by this method to take a syringe of blood from the recipient and put it direct into the donor. Prompt treatment by penicillin and a sulphonamide would be indicated.

DELAYED REACTIONS

In blood transfusions our chief concern is that the donor's cells (containing agglutinogens) shall not be agglutinated by the recipient's serum (containing agglutinins).

If a group "O" donor gives blood to a recipient who is not group "O" the donor's plasma may agglutinate and hæmolyse some of the recipient's cells and cause delayed jaundice. This, however, is not serious because the donor's plasma is small in amount and its agglutinins of low titre. Also, all the cells of the recipient's tissues, and not only his red blood corpuscles, absorb the transfused agglutinin.

Similarly, if a patient who belongs to the 15 per cent whose blood corpuscles do not contain Rh agglutinogens (*i.e.* who are Rh negative) is transfused with Rh positive red cells or becomes pregnant with an Rh positive foetus, she will produce Rh antibodies. Subsequent transfusion with Rh positive blood will be followed by hæmolysis and jaundice. Since 85 per cent of blood is Rh positive, the risk is considerable, and blood selected by the ordinary "A,B,O" compatibility tests is very likely to be incompatible in Rh negative patients. These risks do not apply to Rh positive recipients since anti Rh agglutinins do not occur.

naturally and only appear as a result of transfusion or pregnancy Rh negative blood should therefore be used to transfuse Rh negative patients, particularly for second transfusions, and for pregnant women who have been previously transfused

Group "O" does not simply mean that "A" and "B" agglutinogens are absent "O" is a positive factor and may act as an antigen, and so when the same group "O" donor is used more than once, his cells immunise the recipient against "O" agglutinogens and reactions may occur

True transfusion reactions should be reported to the blood supply depot concerned A few c cms of blood should be left in the bottle and kept after every transfusion lest a reaction should occur and investigation be wanted

C ALLAN BIRCH

CHAPTER IV

2. *Acute (Non-Surgical) Abdominal Catastrophies*

THE four main features of an abdominal emergency are pain, rigidity, vomiting and distension. All of these have well recognised "surgical" causes, but each may be produced by a medical condition and mislead the surgeon into undertaking an unnecessary laparotomy. A helpful way of dealing with the subject is to consider each symptom or sign in turn, noting what medical condition may cause it and how it may be distinguished from the same symptom or sign attributable to a "surgical" cause. Each condition will have to be considered in some detail, since the physician's problem is not that of the surgeon, namely, whether to operate or not. While in most of the conditions which follow, acute abdominal pain is present, it is rarely persistent. *The old rule still holds that the majority of abdominal pains lasting longer than six hours are of surgical nature.*

It is important to be on guard against the presence of an acute medical and surgical condition at the same time. Thus pyelitis and pneumonia may occur with appendicitis, and coronary occlusion with cholecystitis. Another source of trouble is the patient who is known to have had coronary thrombosis and who later has another somewhat similar attack which turns out to be a perforation.

Errors constantly occur because the surgeon neglects to use the stethoscope, or the physician the finger stall. No method of bedside examination is the prerogative of any one kind of doctor. Remember how Osler defined a consultant—"a man who makes the rectal examination after the other physicians passed it up."

The medical "acute abdomen" is not a rarity, and has been found to account for 15 per cent. of cases presenting abdominal symptoms. More than one of the cardinal signs are usually present but one is outstanding. In this account we shall be concerned almost entirely with differential diagnosis.

PAIN

Abdominal pain causes most of our difficulties and is of two main types visceral and peritoneal according to its origin

Visceral pain is caused by distension or spasm of a viscus. It is diffuse it comes and goes and is unassociated with rigidity or pyrexia. Tenderness is minimal. Often the patient is restless. Biliary colic is a good example, while of the extra abdominal causes coronary thrombosis produces many of the features.

Peritoneal pain is caused by irritation of the peritoneum. It is localised constant and associated with rigidity and often with pyrexia. Tenderness is marked and the patient lies still. Perforated gastric ulcer causes this type of pain which also occurs as a reflex phenomenon in pleurisy.

In addition to pain of which the patient complains spontaneously we must also mention tenderness or pain elicited on palpation. A useful point in the differentiation of tenderness caused by some extra abdominal condition is that in true peritonitis there is definite tenderness of the pelvic peritoneum on rectal or vaginal examination.

Tenderness is very important in children and if a conscious child makes no attempt to remove the palpating hand the condition is probably not a local one such as appendicitis.

Coronary thrombosis

Coronary thrombosis with abdominal pain is a constant source of difficulty in its differentiation from a perforated ulcer. A history suggestive of coronary disease is more helpful than one of indigestion particularly if it can be established that there has been pain brought on by exertion and if the present acute pain *begins* behind the sternum rather than in the abdomen. A clear history is however often unobtainable and we are faced with a patient who has sudden severe overwhelming epigastric and retrosternal pain. Usually by the time we see him radiation has occurred. If this is up into the chest and down the arms it is in favour of coronary thrombosis while radiation over the abdomen favours perforation. Shoulder and scapular pain especially on the right side is common after perforated duodenal ulcer with a subdiaphragmatic collection of fluid.

Other factors help us to decide. The pulse in perforation is slow at the onset whereas it is rapid in coronary thrombosis. *Respiration is shallow and thoracic in perforation but abdominal*

in coronary thrombosis. Rigidity in perforation other than into the lesser sac, is extreme and persistent, whereas the rigidity caused by cardiac infarction is not boardlike and varies with respiration. Circulatory failure from the shock of perforation soon passes off, but in coronary occlusion it steadily dominates the picture. In perforation, escaped gas causes the liver dullness to disappear. As distended bowel may prove confusing by also obscuring the normal liver dullness, an X ray film is often of great help by revealing an air bubble under both domes of the diaphragm. Leucocytosis, pericardial friction and signs of congestive failure appear later, and so are of no help at the onset of the emergency. Special consideration should be given to the following points —

- (1) The fall of blood pressure is greater and more sudden in coronary thrombosis than in the shock of an acute abdominal catastrophe. If the patient's previous blood pressure is known this sign may be very significant.
- (2) Arteriosclerosis of peripheral and retinal vessels is somewhat in favour of coronary thrombosis.
- (3) Dyspnoea especially of Cheyne Stokes type suggests a cardiac cause.
- (4) The response to morphine. Though morphine will relieve the pain of intra abdominal disease it acts best when the cause is extra abdominal.
- (5) The electrocardiogram. Although not invariably present in the early stages of an acute myocardial infarction the Pardee curve can on occasion be diagnostic even within an hour or two of the onset. The feature indicative of myocardial necrosis is displacement of the S T segment. The T wave originates directly from the downstroke of R in lead 1 and the upstroke of S in lead 3 (anterior infarction). The opposite pattern depression of S T in lead 1, with elevation in lead 3, is produced by a posterior infarct. More pronounced deformities may be observed in direct precordial leads. A normal curve does not exclude a recent acute coronary occlusion. Abnormalities of the E C G, which are the permanent results of past trouble and unrelated to the present illness, must not be misinterpreted. Nor must it be forgotten that biliary colic may itself produce temporary changes in the electrocardiogram.

Dissecting aneurysm of the aorta.

This is rarely diagnosed accurately before death. Though it may cause symptoms suggesting both perforation and myocardial infarction, points which should arouse suspicion are that pain is widely distributed, often radiating down both legs, the blood pressure remains high after the attack, and evidence of poor blood supply to the legs appears. Although shock may be profound, the abdominal signs are never quite like those of a major perforation. Liver dulness is not diminished, and X rays show no gas bubble under the diaphragm. If there has been a previous attack an excessively calcified or 'double aorta' may be seen on the radiograph.

Ruptured abdominal aneurysm or aorta.

This causes sudden severe abdominal pain and collapse with early distention and rigidity which may easily be mistaken for perforation. Restlessness is a marked feature. If the doctor is forewarned by the knowledge that the patient had an aneurysm he is fortunate. The condition is nearly always fatal in a short time and before arrangements for laparotomy can be made.

Angina abdominis.

Here attacks of widespread abdominal pain without tenderness are associated with a rise of blood pressure in arteriosclerotic subjects. A tablet of glyceryl trinitrate (gr $\frac{1}{100}$) under the tongue brings rapid relief and offers a therapeutic test of the diagnosis.

Mesenteric vascular occlusion.

It is probable that spasm and thrombosis of branches of mesenteric arteries or veins in old people account for some of the attacks of abdominal pain which pass off without operation.

When the superior mesenteric artery or vein is obstructed by an embolus or thrombus intense abdominal pain results. It is central and continuous, exacerbations are characteristic and they are often associated with vomiting. Distension appears next but there is no rigidity and little tenderness until later when peritonitis complicates the picture. Indeed the contrast between the severity of the symptoms and the paucity of the physical signs is diagnostic. A previous history or a source of embolism such as mitral stenosis, aortic valve disease, or coronary infarction,

makes the diagnosis easier, as does melæna—spontaneous or after enema on rare occasions

Apart from minor cases the prognosis is almost hopeless without operation and resection of the involved portion of the intestine, though a success following treatment with heparin has been reported. The condition is mentioned here because it has medical and surgical aspects and the cardiac condition should be recognised and treated

Congestive cardiac failure.

This quite commonly presents with right hypochondriac pain and vomiting leading us to suspect cholecystitis or a "quiet" perforation. Careful examination should reveal other evidence of cardiac failure. Sometimes there is a history of attacks of right hypochondriac pain following exertion, and presumably caused by liver distention

Acute pancreatitis.

This condition is placed among the medical emergencies because better results are obtained by observation and the use of morphine than by operation. This is especially true when acute pancreatic œdema rather than true inflammation is present

The patient is usually a middle aged obese subject, often with a history of gall stones. There is very severe epigastric pain and tenderness but not the abrupt onset or board like rigidity of perforation. Shock is marked. Vomiting is profuse and diarrhoea may occur. Slight icterus and a tinge of cyanosis are often present. Occasionally death takes place within an hour of the onset

The following tests are helpful —

- (1) An X ray film of the abdomen showing absence of the gas under the diaphragm which occurs in perforation
- (2) Loewi's test. Examine the pupils. Into one conjunctival sac instil four drops of solution of adrenaline hydrochloride. Wait five minutes. Instil four more drops and wait 30 minutes and then re examine. Dilatation of the pupil is suggestive of acute pancreatitis
- (3) Estimation of diastase in the urine. This is in great excess in acute pancreatitis

It is fair to say, however, that absolute reliance cannot be put on these tests and in the very acute type of pancreatitis

confusion with other conditions notably perforation makes laparotomy advisable. Some would regard these tests as so unreliable as to render valueless any diagnosis of acute pancreatitis not proved by laparotomy or necropsy. A rare exception to this view would be the case in which subcutaneous fat necrosis is present.

Pneumococcal peritonitis

This is most often seen in children and is dealt with on page 254.

Ascites

In rare instances this is mysteriously fulminating and associated with diffuse abdominal pain. It is usually possible to demonstrate free fluid in the abdomen. Pyrexia and increased pulse rate are absent so that observation and peritoneal aspiration to determine the nature of the fluid are justifiable. True peritonitis may be present as the concluding event in a series of tappings in hepatic cirrhosis.

Occasionally in a known case of tuberculous peritonitis acute symptoms may develop apart from those of an obstructive nature. They may be caused by a sudden increase in the number of tubercles and will settle down when treated expectantly.

The diabetic "acute abdomen"

Occasionally a diabetic passing into coma has severe epigastric pain possibly from distension of the liver by fat. If the patient is not a known diabetic the resemblance to a surgical acute abdomen may be very deceptive. Even when urine examination has revealed the presence of ketone bodies an acute surgical condition cannot be excluded by this finding since it may co-exist and if it be inflammatory it may have been the precipitating cause of diabetic coma. Nor will a white blood count be helpful since leucocytosis is common in diabetic coma. A history of anorexia, nausea and drowsiness before the onset of pain is in favour of diabetic coma.

The need for differentiation is not very urgent. We have to decide between ketosis only and ketosis plus, for example, perforation. Operation should not be undertaken until ketosis is yielding to treatment. If the signs and symptoms are still

present when ketosis has disappeared operation should be undertaken (*See also page 203*)

Pyelitis (*See also page 235*)

Acute pyelitis with pyrexia and pain in the right side of the abdomen may resemble appendicitis but pyelitis usually causes a higher temperature and greater leucocytosis. Examination of the urine reveals pus and organisms. Since these are almost normal findings in the ordinary specimen of urine in many women care should be taken in assessing their significance and a catheter specimen obtained. Occasionally urinary abnormalities may be absent because of blockage of a ureter. In such cases cystoscopy will help by showing an acutely inflamed ureteric orifice and catheterisation will bring rapid relief of pain and pyrexia.

Great attention should be paid to the march of symptoms. Mid line pain—vomiting—right sided pain—fever—in this order are strongly suggestive of appendicitis. The absence of right sided pain (of peritoneal origin) should not exclude appendicitis since the organ may be retro caecal or pelvic. Nor should dysuria necessarily be interpreted as a symptom of pyelitis since pelvic appendicitis may cause it.

It is said that the blood sedimentation rate is more likely to be raised in pyelitis than in unruptured appendicitis and this test may be helpful in a doubtful case.

Acute non specific mesenteric lymphadenitis (Brennemann's syndrome)

This condition occurs especially in children and causes symptoms resembling appendicitis. While differentiation may not be sufficiently clear to justify expectant treatment certain points of difference should be noted. In lymphadenitis the tongue is cleaner and the temperature higher than in appendicitis. Pain is often severe and colicky and usually central. Tenderness also is more in the mid line than in the right iliac fossa. Shifting of the point of maximum tenderness from altered direction of pull on the mesentery when the patient lies on the left side for a time suggests adenitis. Vomiting occurs earlier than in appendicitis. Both conditions may be causally related to throat infections. Attacks with complete remissions are usual. When symptoms

persist tuberculous adenitis should be suspected and calcification looked for by X ray examination. Glandular enlargement of known origin (e.g. glandular fever) is not usually associated with acute symptoms.

If differentiation from appendicitis is not clear the abdomen should be opened. Otherwise expectant treatment should be adopted. Occasionally the appendix has already been removed.

Acute hæmoperitoneum (*See also Mittelschmerz, page 82*)

This may result when the amount of blood lost from a ruptured Graafian follicle is more than the usual minimal amount—so called apoplectic ovary. There is sudden lower abdominal pain with nausea but without pyrexia or a coated tongue. Tenderness is found on vaginal examination. An attack occurs typically midway between the menstrual periods. Symptoms disappear within a day or two. A similar picture may develop at any time if an ovarian blood cyst ruptures or a necrotic metastasis bleeds or an aneurysm leaks. As the symptoms are more severe in these cases surgical intervention is usually necessary.

General infections and toxæmias

Abdominal symptoms may occur in the course of known infectious illnesses and suggest that some other condition is also present. We should be chary however of diagnosing an acute abdominal condition when general symptoms are present. It has been said that an acute abdomen with a headache is never an acute abdomen.

TONSILLITIS in children is often associated with abdominal pain and sometimes with true appendicitis. Appendicular pain and tenderness over the appendix may occur in the prodromal stage of SCARLATINA and MEASLES. But true appendicitis is not part of the picture of these diseases. A white blood cell count and a search for Koplik's spots are useful aids in diagnosis. PANCREATIC MUMPS causes pain and vomiting but no rigidity and if parotid mumps is subsiding diagnosis is easy. A history of contact is suggestive.

IN ACUTE RHEUMATISM severe lancinating abdominal pain may occur. It is worse on movement and associated with tenderness. A history of acute rheumatism or chorea is usually obtainable.

Vomiting is a common symptom of MALARIA and especially

malignant tertian infections in fulminating cases with visceral lesions there may be acute abdominal pain but there is no rigidity.

In abdominal INFLUENZA there may be griping abdominal pain in addition to headache and pharyngitis. Rigidity is absent and the extra abdominal symptoms suggest the diagnosis.

Acute diseases of the spinal cord (*e.g.* ACUTE POLIOMYELITIS) or nerve roots (*e.g.* ZOSTER) may begin with abdominal pain. A period of watching will reveal typical signs (paralysis or a rash) of the true nature of the illness.

In rare cases when the small causal wound has been unrecognised abdominal rigidity and pain have been the presenting signs of TETANUS. Toxins of industrial origin such as LEAD (*page* 317) and CARBON TETRACHLORIDE (*page* 317) must be remembered as causes of acute abdominal pain. It must not be forgotten that lead may be ingested in many unusual ways. The bite of certain SNAKES (*page* 301) in the southern states of the U.S.A. has produced severe abdominal pain and rigidity of the abdominal wall.

Abdominal pain and vomiting are occasional manifestations of idiosyncrasy to OPIUM and MORPHINE usually in large doses and especially in women. The mechanism is probably intense spasm of involuntary muscle in the biliary tract or elsewhere.

INFECTIVE HEPATITIS may give rise to pain and tenderness in the right upper abdomen with vomiting and moderate pyrexia resembling appendicitis. Careful study of the march of events and a search for bile in the urine and slight jaundice of the skin and conjunctivæ should point to the correct diagnosis.

TYPHOID may be confused with appendicitis particularly in sporadic cases in children. The immediate diagnosis in an isolated case presenting with acute pain in the right iliac fossa would be a matter for congratulation. Very few of these patients escape appendicectomy but if the possibility of typhoid is borne in mind the demonstration of a leucopenia may stay the surgeon's hand. A perforated typhoid ulcer may be very silent at first and present a little later as a medical emergency when gas under the diaphragm (as seen on X-ray examination) causes respiratory symptoms and spreading peritonitis creates the usual clinical picture.

Acute gastro-enteritis and ileo colitis

The diarrhoea and vomiting of gastro-enteritis may cause

diffuse abdominal pain and tenderness. Care must be taken to exclude appendicitis, for it is too often forgotten that an inflamed appendix in the pelvis may itself cause diarrhoea. In a child the pain, tenderness and rigidity of appendicitis may be slight, and hence the need for great care in differentiating appendicitis from gastro-enteritis. Special attention should be paid to tenderness since it is a much more important feature of appendicitis than pain.

Ileo colitis in children resembles intussusception because in both conditions there is colicky pain with the passage of bloody mucus. The essential features of intussusception are described on page 253. Here, only the differential points are described. Passage of pure blood and clots does not occur in intussusception except in the rare case where an ulcerated Meckel's diverticulum forms the apex of the intussusception. Symptoms are less acute when the colon rather than the ileum forms the apex. Pyrexia, excoriation of the buttocks by evacuated succus entericus and the peculiar smell which some can recognise are in favour of ileo colitis. Prolonged abdominal palpation, especially during a paroxysm, is important, and absence of the typical sausage shaped tumour which contracts and relaxes is in favour of ileo colitis. Microscopical examination of the faecal mucus shows numerous pus cells and a dysentery bacillus can often be grown on culture. The child with an intussusception is plump and healthy looking by contrast with the usual case of ileo colitis and it is remarkable how well he may appear between the paroxysms. Indeed after the first sudden severe bout of pain the subsequent attacks may be relatively mild.

Henoch's purpura. (*See also page 142*)

In this condition pain is colicky and caused by spasm of the bowel and infiltration of its wall with blood and serum ('visceral hives'). Rigidity, vomiting and distension are also present. Abdominal pain nearly always precedes the appearance of skin purpura and until the latter appears the diagnosis may be in doubt. Joint pains and albuminuria are helpful differentiating points, and purpura induced in the arm by applying a tourniquet (Rumpel-Leede test) is suggestive. A past or family history of allergy should be sought. Even when skin lesions make the diagnosis certain it must not be forgotten that Henoch's purpura

is not entirely a medical disease and that surgical complications such as intussusception may arise

Allergy as a cause of acute abdominal symptoms

Allergic abdominal symptoms are similar to those described under Henoch's purpura. Often there is a history that the attack began after eating some food such as pork to which the patient was known to be sensitive. If urticaria appears the diagnosis is confirmed. The blood picture usually shows leucopenia with eosinophilia. Adrenaline by injection will alleviate the symptoms promptly. Cases have been reported of recurrent attacks of severe abdominal pain with pyrexia and signs suggesting appendicitis. Laparotomy in one such case revealed only great congestion of the subserous vessels.

Bornholm disease

This condition (also known as Epidemic Pleurodynia and acute benign dry pleurisy) presents with pain of pleuritic character as the principal symptom. When this is abdominal and pleural friction absent an acute abdominal emergency may be simulated.

This should be excluded by the presence of other features of Bornholm disease such as headache and myalgic pains elsewhere. Tenderness of the costal margin and leucopenia. When the disease is epidemic the diagnosis is easy.

Visceroptosis

While true ptosis of abdominal viscera may be a cause of disability the symptoms are usually of a chronic nature. Acute pain and vomiting are almost invariably attributable to the associated constitutional and nervous element. The patient complains of agonising pain for which examination reveals no adequate explanation. Indeed examination may be difficult on account of the commotion that the patient creates. Experience of these cases makes them easily recognisable. Morphine abolishes the symptoms completely but it is useful to give first 1 c.c. of sterile water and record the effects of this placebo after 15 minutes. It may then be found that morphine is unnecessary. Nevertheless such patients may develop appendicitis and other acute diseases and if there is localised tenderness great care should be exercised and a leucocytosis sought.

Acute hæmatoporphyria.

In this very rare condition acute lower abdominal colic occurs. Vomiting and the absence of diarrhœa suggest obstruction. The urine is of a port wine colour and contains iron free pigment (porphyrin) derived from hæmoglobin. When it is acidified and examined under ultra violet light a pink fluorescence is seen.

RIGIDITY

Abdominal rigidity of medical origin is usually caused by disease of the lungs and pleura particularly in children (*page 254*). It is more a stiffness of the abdominal wall than the board like rigidity of perforation and some prefer to call it resistance. When vomiting is an early symptom as in a child the resemblance to true abdominal disease is great. The only medical condition causing real board like rigidity is the spasm of tetanus.

Some unequivocal evidence of chest mischief such as a pleural rub or bronchial breathing often settles the matter. Slight cyanosis, inspiratory dilatation of the nostrils, raised respiratory rate and the absence of rectal tenderness all point to the chest as the site of the trouble. Reflex rigidity caused by pleurisy relaxes during breath holding. It is not associated with restriction of respiratory movement of the abdominal wall. Leucocytosis is higher in the initial stage of pneumonia than it is in appendicitis before perforation occurs.

Rigidity may be very marked in spontaneous pneumo thorax and particularly hæmo pneumo thorax. Typical signs of pneumo thorax (resonance with silence or distant amphoric breathing) or of air and fluid in the chest (splashing) are generally present. In a doubtful case the chest should be X rayed. The rigidity of extra abdominal disease is a response to pain and being central in origin it diminishes after morphine. Rigidity in intra abdominal disease persists after morphine.

Diaphragmatic pleurisy.

This especially provides a pitfall for the unwary for it causes referred pain in the abdomen and resistance. If the central portion of the diaphragm be involved pain may be referred to the shoulder tip. Physical signs are disappointingly few but lack of movement of one side of the chest and activity of the

alæ nasi should point to the chest as the seat of the trouble. A rare but helpful symptom of pleurisy is persistent and painful hiccough. Other points in favour of pleurisy are a flushed face with a tinge of cyanosis, herpes of the lips and a history of an initial rigor. Again the march of events should be carefully scrutinised for the patient with diaphragmatic pleurisy or pneumonia is too sick, too soon to have appendicitis. In rare cases an acute primary diaphragmitis produces a similar picture but without evidence of pulmonary disease.

Hæmatoma of the rectus muscle

This condition may cause localised hardening of the tissues simulating rigidity. It may occur spontaneously in debilitated patients. The lower part of the rectus abdominis muscle has no posterior sheath and a hæmatoma there may cause peritoneal irritation. Discoloration of the skin and the presence of a superficial lump point to the correct diagnosis.

VOMITING

When vomiting is the main symptom and pain is absent or atypical a medical cause is likely. Difficulty chiefly arises when vomiting is associated with pain and distension also of medical origin. It must be emphasised that *pain with vomiting* is characteristic of small bowel obstruction which should be diagnosed before the distension appears. Hence very great care must be taken before deciding that there is no obstruction.

Tabes dorsalis

The vomiting of gastric crises in tabes is a classical pitfall in diagnosis. Usually there is more retching than vomiting. Careful questioning will show that the epigastric pains have the same characteristics as those elsewhere, namely they are lancinating and occur in rapidly recurring paroxysms. In an uncomplicated tabetic crisis abdominal rigidity is absent. It is not sufficient to test the knee jerks alone since a tabetic crisis can occur when knee jerks are normal if the dorsal rather than the lumbar posterior roots are mainly involved. The ankle jerks should be examined and analgesia to pin prick sought.

Acidosis

Ketone bodies may be found in the urine of any child who has vomited frequently. They are the *result* and not the cause of vomiting whether this be medical or surgical. The

picture of cyclical vomiting is that of fever vomiting and pale stools in a child who is highly strung and has had a diet containing excess fat. Often some infection such as tonsillitis starts off the attack. Rigidity is absent and pain is probably caused by straining of the abdominal wall by the vomiting which precedes it. A careful study of the march of events will indicate that the cause of the ketosis is not surgical.

Acute poisoning

The doctor should always keep at the back of his mind the possibility that the cause of the symptoms may be poisoning or at any rate some ingested irritant. Patients nearly always try however to attribute their abdominal symptoms to some dietary indiscretion and therefore we must be on our guard against accepting their explanation too readily and missing a true appendicitis. Poisoning by bacteria and their toxins, inorganic poisons, green apple colic and acute alcoholic gastritis may all cause acute abdominal symptoms. Bolus colic may be suspected if a patient vomits a mass of obviously indigestible material.

Pernicious anæmia

Although severe pernicious anæmia is becoming rare, cases with abdominal crises of pain and vomiting are occasionally seen. The pain is biliary colic from the passage of bile mud produced by a hæmolytic crisis. The profound anæmia with lemon tint, smooth tongue and paræsthesiæ should make one cautious and seek confirmatory evidence in a blood examination. Retinal hæmorrhages are an additional diagnostic feature.

Acholic jaundice

Abdominal crises similar to those of pernicious anæmia may occur in acholic jaundice. There is abdominal pain and vomiting. Sometimes tenderness is discovered and suggests intra-peritoneal hæmorrhage. A history of jaundice and of previous attacks should make the diagnosis clear.

Uræmia

Uræmia or chronic renal failure is a common pitfall for the surgeon. The intense vomiting of sudden onset mimics high intestinal obstruction. The resemblance is greater if the patient is very thin so that normal peristalsis is visible. The vomit is not fæculent as it may be in obstruction. Hæmatemesis from uræmic gastritis or from bleeding gums may confuse the issue.

The general toxic appearance the dry, furred tongue, and evidence incriminating the kidneys such as a history of nephritis and hypertension, together with the presence of urinary abnormalities will indicate the correct diagnosis. In congenital cystic disease with uræmia the kidneys are palpable. Peristaltic sounds are not increased in uræmic vomiting as they are in obstruction.

Sometimes the acute terminal pericarditis of uræmia causes upper abdominal pain, and the clinical picture closely resembles that of perforation.

Other medical causes of severe vomiting which may cause confusion are migraine (*page 179*), Meniere's syndrome (*page 181*), and the crisis of Addison's disease (*page 222*). Should the eye condition be overlooked the vomiting of glaucoma may be misleading.

DISTENSION

When this is of recent origin and is associated with vomiting it indicates intestinal obstruction which incidentally, should have been diagnosed at the stage of yellow brown vomit before distension appeared. The hernial rings must not be forgotten. Abdominal distension may be a marked feature of acute hydronephrosis.

Occasionally great distension may complicate the picture of some other disease such as typhoid, pneumonia or ulcerative colitis and make one wonder if a "surgical" cause is present. If the original diagnosis is firmly based no difficulty should arise.

A rare condition not very well known or understood is that known as 'bloating' or abdominal distension not due to gas. The distension is not present in the morning but gradually appears as the day goes on and may vanish suddenly without the passage of flatus. Lying on the back and flexing the thighs will cause it to disappear, as will a dose of morphine. It is quite distinct from pseudocystitis, and appears to be caused by descent of the diaphragm and cramp-like contraction of the abdominal muscles. It occurs in psycho-neurotic women.

Tight distension or rigidity plus distension is a late picture and indicates intestinal obstruction plus peritonitis. A rare cause of great abdominal distension in a young baby is an ano-rectal stricture. The distension is relieved by passing a catheter into the bowel, and cured by dilatation with the finger.

CHAPTER V

Other (Non-Surgical) Abdominal Emergencies

IN this chapter will be considered acute conditions affecting the abdominal organs which do not as a rule simulate acute surgical conditions and which are not obstetrical or gynaecological. These are —

- Vomiting
- Acute food poisoning
- Biliary colic
- Hepatic failure and the hepato renal syndrome
- Rectal pain
- Priapism
- Acute lumbago and
- Gastro duodenal hemorrhage

VOMITING AS AN URGENT SYMPTOM

Vomiting is a common and sometimes a presenting symptom in many urgent illnesses. The following classification is therefore given as an aid in reviewing possible causes in an obscure case. Obvious causes such as post anaesthetic vomiting are not included. Usually some associated symptom or sign such as headache or papilloedema will point to the diagnosis.

CAUSES OF VOMITING

- 1 Those acting on the vomiting centre
Many acute infections (e.g. Pneumonia) and acute specific fevers (e.g. Scarletina). Crisis of Addison's disease
- 2 Those acting reflexly
 - (a) Through the stomach itself
 - Irritant poisons and unsuitable food
 - Organic disease of the stomach
 - In children—congenital hypertrophic pyloric stenosis and habit vomiting

- (b) Through the abdominal and thoracic viscera
 Appendicitis Peritonitis Intestinal obstruction
 Violent coughing from any cause
- (c) Through the nervous system
 - 1 The brain Increased intracranial tension from any cause Meningitis Migraine
 - 2 The spinal cord Gastric crises of tabes dorsalis
 - 3 The special senses Meniere's syndrome Sea sickness Unpleasant sights and smells

Treatment of vomiting depends on its cause and unless this is dealt with, symptomatic measures such as drop doses of weak solution of iodine dilute hydrocyanic acid cerum ovalate and iced champagne are not likely to be effective Gastric aspiration and lavage by the nasal route are temporary expedients in intractable cases (uræmia) or in those cases of unknown etiology

ACUTE FOOD POISONING

General management.

Since this may be a communal as well as an individual emergency it is important to realise its implications at the onset and to act before evidence is lost

- (1) Make a note of the foods eaten prior to the attack The following classification will help in deciding which food to suspect

BACTERIAL FOOD POISONS —Symptoms within two to four hours after eating the food, which is commonly cream cake or trifle containing staphylococcal toxin

BACTERIAL FOOD INFECTIONS —Symptoms more than 12 hours after eating the food, which is commonly cream cake or trifle (dysentery organisms) or manipulated meats and duck eggs (salmonella organisms)

POISONOUS FOODS, e.g. —Fungi taken in mistake for mushrooms
 Belladonna leaves accidentally included in dried herbs
 Narcissus bulbs eaten in mistake for onions
 Rhubarb leaves used as spinach

Foods sometimes poisonous include potatoes (especially if sprouting), mussels and cheese

TOXICOLOGICAL CHEMICAL POISONS—Salts of lead zinc arsenic and antimony may contaminate tinned foods or may be intentionally added to any food

- (2) Take charge of suspected food—not forgetting left over scraps
- (3) Find out where the food came from and report to the medical officer of health so that he can send an inspector there at once
- (4) Save a specimen of vomit urine and feces for laboratory examination Taking a rectal swab may save time if feces are not available

Symptoms—These are vomiting diarrhœa and colicky abdominal pain In shell fish poisoning urticaria is common Circulatory failure may be the main symptom in some cases

Treatment—If vomiting is troublesome the stomach should be washed out with a gallon of warm water containing a table spoonful of sodium bicarbonate After gastric lavage give castor oil 1 fl oz for an adult with orange juice if desired A hot water bottle on the stomach is comforting Nikethamide 2 c cm by injection may be needed

When vomiting has ceased give fluids fruit juice diluted with half strength saline by mouth Some prefer effervescent drink in small amounts In collapsed patients 5 per cent dextrose in normal saline may be given intravenously Leave a prescription for bismuth and opium

R Tincture of opium m 10 Sodium bicarbonate gr 15
Bismuth carbonate gr 30 Chloroform water to 1 fl oz
To be taken every four hours

Morphine is best withheld until vomiting and diarrhœa abate

Tenesmus

This means the urgent desire to empty the rectum the act being accompanied by straining It is a distressing symptom in some cases of diarrhœa It may be caused by severe enteritis impacted feces or even a foreign body If rectal examination is negative a starch mucilage enema with opium as a palliative measure may be tried A heaped tablespoonful of starch is mixed with two tablespoonfuls of cold water to form a smooth paste To this is added while stirring one pint of boiling water After allowing to cool 4 to 6 fl oz with 30 minims of tincture of opium are given slowly as an enema at body temperature

Botulism

This rare emergency is considered here because it is a form of food poisoning although its symptoms are nervous and not abdominal

The causal organism and its toxin are destroyed by heat but the spores are not. They develop anaerobically and produce toxin in canned meat foods which then have an offensive odour. The organism does not produce toxin in the intestine.

Symptoms—diplopia, dysphagia and muscular weakness—result from the effects of toxin on the nervous system. Pain and pyrexia are absent; the victim remains mentally alert. Slight initial nausea and vomiting caused by proteolytic products may occur.

Treatment consists of administering antitoxin but as this must be the correct type, laboratory help is essential in its use. The Ministry of Health, Whitehall, S.W. 1 (Tel. Whitehall 4300) or The Department of Health for Scotland, St. Andrew's House, Edinburgh (Tel. Edinburgh 34433) should be consulted. Ether anaesthesia and alcoholic beverages are said to delay fixation of toxin in the tissues.

“MUSHROOM” POISONING

This is considered in detail because specific treatment is available. It is uncommon—only 38 fatal cases having occurred in England between 1920 and 1945—and so its very serious hazards are apt to be forgotten.

Many brilliantly coloured and peculiarly shaped fungi are not poisonous at all or are merely indigestible. They are generally avoided. The very poisonous Death Cap (*Amanita phalloides*) however is easily mistaken for the edible mushroom.

Tests of edibility

Most of the popular tests are fallacious. The edible mushroom peels easily but so does the Death Cap. Some fungi change colour alarmingly when cut but this does not indicate that they are poisonous. Failure to blacken a silver spoon during cooking is no proof of edibility. The fact that fungi are eaten by rabbits does not mean that they are safe for man as rabbits can eat the Death Cap with impunity. Hence it is only by recognising poisonous fungi on sight that emergencies can be avoided.

The Death Cap (*Amanita phalloides*) (Fig 7)

The cap of this fungus is smooth and yellowish green but is subject to considerable variation in colour. The gills however

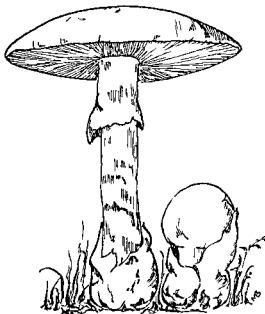


FIG 7

The Death Cap (*Amanita phalloides*) Gills white volva at base of stem cap yellowish green
(Height $4\frac{1}{2}$ in width of cap $3\frac{1}{2}$ in width of stem $\frac{3}{4}$ in) (Prattitioner)

are permanently white and this is an important distinguishing feature as the gills of the edible mushroom are never white (Fig 8). The base of the stem of the Death Cap is surrounded by a persistent cup or volva but this may be missing if the whole fungus is not gathered. No volva is present on the stem of the edible mushroom. Death Cap spores are identifiable as white sub globoid bodies measuring 8 to 11 by 7 to 9 μ with a central oil drop.

The relative measurements of the Death Cap and the edible mushroom are (in inches) —

	Height	Width of cap	Width of stem
Edible mushroom	$3\frac{1}{4}$	5	1
Death Cap	$4\frac{1}{2}$	$3\frac{1}{2}$	$\frac{5}{8}$

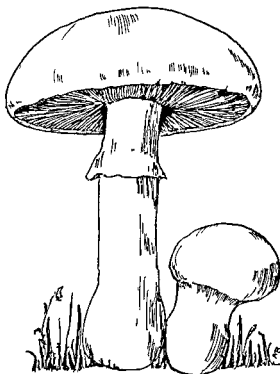


FIG 8

Edible mushroom (*Psalliota campestris*) Gills brownish purple base of stem clubbed no volva
(Height $3\frac{1}{4}$ in width of cap 5 in width of stem 1 in) (Practitioner)

Symptoms—From the point of view of treatment cases of “mushroom” poisoning fall into two groups—(1) Those with early symptoms (2) Those with delayed symptoms

Early symptoms—These are diarrhoea and vomiting and may be caused by many fungi and even edible ones if not fresh, or if eaten by allergic subjects Sometimes the early symptoms are

nervous—delirium and hallucinations—as from eating *Amanita muscaria* (Fig 9)



FIG 9

Fly Agaric (*Amanita muscaria*) Cap red with white patches (Height 10 in width of cap 7 in width of stem 1 in) (Iractitioner)

Delayed symptoms—These come on after a latent period of eight hours or more and this delay is characteristic of Death Cap poisoning. After this interval, vomiting and diarrhoea with intense abdominal pain begin. These symptoms may abate a little, but collapse and death occur in at least half the cases from liver function failure or cholæmia.

Treatment—The decision whether to treat for Death Cap poisoning or not may be made by—

- (1) Examining the uneaten fungi
- (2) Considering the time interval between ingestion and symptoms
- (3) Examining the vomit and fæces for spores

If in doubt treat for the more serious condition as follows—

- (1) Give atropine gr $\frac{1}{10}$ intravenously to an adult This is the antidote to muscarine like alkaloids Nikethamide 2 c cm repeatedly may be needed for collapse
- (2) Wash out the stomach and leave in it 2 fl oz of White Mixture or a 50 per cent saturated solution of Epsom salt Take special care to keep the head lowered if the patient is comatose so as to avoid inhalation of gastric contents This treatment will probably be too late in Death Cap poisoning but it would be wise to consider its use in every case
- (3) Empty the bowel by enema
- (4) Start energetic treatment as described under Hepatic Failure (page 57)
- (5) Obtain and administer antiphallenic serum

Apply to The Director Central Public Health Laboratory London N W 9 (Telephone COLindale 6041), or, failing this try The Lister Institute, Chelsea Bridge Road, London, S W 1 (Telephone SLOane 2181 Telegrams Bacteriology, Knights London) or Institute Pasteur 28 Rue de Dr Roux Paris XV (Telephone Segur 01 10) Ask for "Serum antiphalloldien"

The dose will be indicated on the amount sent It is usually about 40 c cm and should be given intramuscularly or intravenously with the usual precautions

If serum has to be obtained from France air transport can be arranged through British Overseas Airways Corporation Airways House London S W 1 (Telephone VICToria 2323 Croydon Airport Telephone CROydon 4422) or London Office of Air France 2b Eccleston Street London S W 1 (Telephone SLOane 0701 Croydon Airport Telephone CROydon 7744 extension 236) In case of difficulty in Paris it is suggested that the British Embassy there be approached at Rue du Fauborg St Honore Paris (Telephone Anjou 27 11) It might be quicker to ask the Scientific Office of the French Embassy in London 1 Carlton Gardens S W 1 (Telephone WHIttehall 5444) to have the serum sent over by means of the diplomatic bag from Le Bourget Airport to Croydon Airport I make no excuse for detailing several routes since I have not had the occasion to try any of them

- (6) Give the rabbit stomach brain treatment of Limousin and Petit

This is based on the fact that while cats die after eating *Amanita phalloides*, rabbits do not But the juice of *Amanita phalloides* injected subcutaneously into rabbits is fatal, suggesting that the toxin is destroyed or neutralised in the rabbit's stomach Cats fed on *Amanita phalloides* plus rabbit's stomach survive for several days If rabbit's brain is given also, they recover completely

The treatment recommended is to give up to five uncooked rabbits brains and stomachs minced up daily for several days. Its practicability in a vomiting patient seems questionable but the emergency is so desperate that it should be tried. All those who have shared the meal containing fungi *irrespective of whether they have symptoms or not* should submit to stomach lavage.

Bowel lavage is of doubtful value since fungi are unlikely to have reached the colon. A more effective measure after gastric lavage and leaving White Mixture B.P.C. in the stomach is to repeat ounce doses of Epsom salt and drinks of hot tea every half hour until the bowels act.

BILIARY COLIC

Biliary colic most commonly results from distension of the biliary tract due to spasm at the ampulla of Vater caused by the impaction there of a gall stone. It is one of the most severe pains known. It is a visceral pain and so is felt centrally at first but soon radiates to the right hypochondrium and right scapular region. It comes and goes at intervals of several minutes and with each paroxysm the patient rolls about and is doubled up in agony and sweats profusely. Vomiting may occur at the end of an attack. The patient is left limp, pale and sweating after an attack lasting from a few minutes to several hours. Similar but less severe pain may be due to widespread spasm in the biliary passages and occurs as a sequel to old standing cholecystitis and chronic cholangitis irrespective of whether the gall bladder has been removed or not.

Examination between the paroxysms reveals a tender liver and resistance of the upper right rectus muscle but little else. Marked local tenderness and rigidity suggest peritoneal pain from cholecystitis. Evidence of slight jaundice—clinical or bio chemical—helps to clinch the diagnosis. When colic complicates acholuric jaundice pigmentation is obvious.

Differential diagnosis

Coronary thrombosis may be simulated (see pages 34 and 127). Acute pancreatitis (page 37) may be an associated condition. Other abdominal colics are usually less severe and have their own characteristic distribution and accompaniments.

Treatment—Morphine is often advised but if used it should be given in full doses i.e. gr $\frac{1}{4}$ to $\frac{1}{2}$ intravenously. Smaller doses tend to increase spasm by vagal stimulation and make matters worse. It is preferable to use atropine gr $\frac{1}{100}$ and dilaudid gr $\frac{1}{32}$ intravenously.

Trasentin 6H (Ciba) intramuscularly (not intravenously) is recommended. It is a synthetic anti-spasmodic similar to atropine in action but with fewer side effects. Ampoules contain 1.7 c.c.m. (1.5 c.c.m. contain 0.075 gm. = $1\frac{1}{8}$ grain) and half to one ampoule may be used. Other remedies of which I have no personal experience for this purpose are aminophylline 0.5 gm. in 20 c.c.m. intravenously, pethidine 100 mgm. subcutaneously and anaesthesia induced by intravenous hexobarbitone.

Heat should be applied locally and a strong carminative mixture may help (Compound Tincture of Cardamom B.P. a dessertspoonful in hot water).

HEPATIC FAILURE AND THE HEPATO RENAL SYNDROME

The concept of failure of liver function leading to cholæmia is not so widely recognised as that of failure of kidney function or uræmia probably because being of shorter duration opportunity for its study is limited. Yet liver function failure is a serious emergency demanding urgent treatment. Many agents may damage the liver and lead to its failure. Once the risk is recognised emergency treatment should be started.

Liver damage leading to failure of function may be expected in —

- (1) Any infective condition of the liver and biliary tract—cholecystitis, amoebic and leptospiral hepatitis, epidemic hepatitis
- (2) Acute hepatic necrosis
- (3) Septicæmia
- (4) Cirrhosis
- (5) Poisoning by drugs and anaesthetics
- (6) Industries in which certain volatile solvents are used (e.g. carbon tetra-chloride)
- (7) Thyrotoxicosis
- (8) The sudden (surgical) release of biliary tract obstruction

Because of the many ætiological factors the symptomatology

will be varied but in a patient suffering from any of these conditions warning of failing liver function is given by lethargy malaise and a dry muddy skin followed by vomiting jaundice and a tendency to hæmorrhage. The imminence of hepatic failure may be heralded by minor alterations of demeanour or behaviour a previously cheerful well behaved patient becomes awkward or truculent for no apparent reason. Such psychological upsets are of supreme importance but their true significance as urgent symptoms is likely to be overlooked by the nurses unless they have been warned in advance. In a patient with a draining gall bladder diminution of the flow of bile often heralds disaster.

By excreting toxins with which the liver is unable to deal the kidneys may be damaged and so oliguria albuminuria and hæmaturia are added to the picture (hepato renal syndrome).

Treatment—The liver has so many functions that it is justifiable to employ various measures calculated to protect each of them.

- (1) Glucose. The liver is protected from the action of toxins when it contains adequate stores of glycogen provided by large doses of glucose. Hypoglycæmia in hepatectomised dogs can be prevented by the intravenous injection of glucose at a rate of 0.25 gm per Kg per hour. A patient in cholæmia may be compared with a hepatectomised animal and if he weighed 70 Kg he would need on the basis of 0.25 gm per Kg 420 gm of glucose in 24 hours. It may be given by mouth and intravenously.
- (2) Insulin aids the deposition of glycogen and should be given in doses of 10 units for every 50 gm of glucose.
- (3) Calcium. Give 10 c cm of 10 per cent calcium gluconate intravenously every four hours.
- (4) Vitamin B. Give 12 000 units (36 mgm) daily by injection.
- (5) Vitamin K. Give 10 mgm daily by injection.
- (6) Blood transfusion. This seems to be the most practicable method at present of increasing the albumin content of the blood which is low in many liver diseases.
- (7) Methionine and choline.

It has been shown that experimental hepatic necrosis can be prevented by an amino acid—methionine—and fatty infiltration by choline. Although there is no evidence that these substances can reverse established changes it would seem reasonable to

consider their use in preventing further damage. Their real value in clinical medicine is still sub judice.¹

Methionine and choline have been given in doses of 6 gm daily both by intravenous drip. Since choline has the same action on the autonomic nervous system as acetylcholine it is advisable to give atropine in doses of gr $\frac{1}{100}$ to $\frac{1}{50}$ with choline to prevent abdominal pain and excessive sweating and bronchial secretion.

"HYPERPYREXIA DEATH"

Following operations particularly on the biliary tract or in patients with damaged livers progressively mounting pyrexia may occur and lead to early death. The mechanism is not clear but some cases at any rate seem to be the result of hyperacute infection. It would seem reasonable to treat this emergency by penicillin and a sulphonamide. The nature of the emergency and the therapeutic measures indicated necessitate an adequate fluid intake (e.g. 8 pints in 24 hours) and careful measurement of the urinary output.

RECTAL PAIN

A night call for the doctor may be occasioned by an attack of boring cramp like pain in the rectum just above the sphincter ani. It begins as a slight pain and works up to a maximum in five or ten minutes. Sometimes it is associated with sexual intercourse. There is no diarrhoea or passage of flatus. A fainting attack may complicate the picture.

Local examination is entirely negative but should be made since similar pain may result from impacted feces.

The condition has been variously called paroxysmal proctalgia, rectal neuralgia, proctalgia fugax and rectal crises of non tabetic origin.

A small warm enema and pethidine 100 mgm by mouth or two compound tablets of codeine B.P.C. will usually cut short the attack.

PRIAPISM

This is a pathological persistent erection of the penis with absence of pain and libido. Erection persists because of thrombosis in the corpora cavernosa. Leukæmia should be suspected.

X ray therapy is the best treatment Dicoumarin (page 439) may be given to prevent further thrombosis

ACUTE LUMBAGO

By acute lumbago we mean a sudden severe low back pain which immobilises the patient His back becomes locked When this follows immediately on a sudden strain such as lifting a heavy object in the flexed position a surgical rather than a medical emergency may be suspected The pain may however appear more gradually after a period of stooping as in gardening and constitute an extremely inconvenient situation if not a dire emergency

Treatment—The patient should be put to bed—often a long and difficult process—and given an analgesic such as three compound tablets of codeine B P C or their equivalent Warmth should be applied in whatever form is most convenient An electrically heated pad to lie on is very comforting If pain is very localised it may be relieved by infiltration of the affected area with 1 per cent procaine containing 0.005 per cent adrenaline hydrochloride

Recurrent attacks or the presence of even slight pain in the distribution of the sciatic nerve make it probable that the locked back is due to a protrusion of an intervertebral disc In such a case the question of more effective early immobilisation than bed rest such as by plaster jacket should be considered

C ALLAN BIRCH

GASTRO DUODENAL HÆMORRHAGE

Recent years have brought radical changes in the management of gastro duodenal hæmorrhage The introduction of liberal feeding and drip blood transfusions have greatly improved the prognosis of bleeding from a peptic ulcer The licking of dry parched lips and the deathly pallor of extreme anæmia are seldom seen in hospital wards to day Nevertheless this emergency is still serious particularly in patients over 50 years and when associated with other complications

Etiology

Peptic ulcer is the most common cause of gastro duodenal hæmorrhage Bleeding may occur from a small superficial acute

ulcer or from a large chronic crater eroding the pancreas and from all gradations between these extremes. All may cause severe and repeated hæmorrhage but while the mortality from an acute ulcer is low (2 per cent) that from a chronic ulcer is as high as 10 per cent. Many acute or subacute ulcers heal quickly and may come and go within a few weeks. Consequently nearly a third of all admissions do not show any radiological evidence of ulcer when examined two to four weeks later. Nevertheless their presence has been shown in many such cases by early gastroscopy. An analysis of 673 consecutive cases of hæmatemesis and melæna showed the following diagnoses—

Peptic ulcer	615
Tumours	23
Portal hypertension	25
Miscellaneous	10
	—
	673

Both carcinoma of the stomach and cirrhosis of the liver are relatively uncommon causes of gastro duodenal hæmorrhage.

Treatment may be discussed under three headings—(1) feeding (2) blood transfusion and (3) surgery.

Feeding

There has been much controversy in recent years on the advisability of liberal feeding. With the traditional starvation treatment there was a mortality rate of about 15 to 25 per cent in all series of a hundred or more admissions. Meulengracht reported in 1934 a 2 per cent mortality in Copenhagen after liberal feeding. Admittedly previous Danish figures showed only a 7 per cent mortality (perhaps there are more acute peptic ulcers in Denmark). Adoption of his recommendations on diet in this country has been accompanied by a fall in mortality which is now between 5 and 10 per cent. The improvement has also coincided with the use of drip blood transfusions which certainly contributed but probably the better figures are due mainly to the elimination of the deaths from dehydration. Nearly half the patients treated by starvation and restricted fluids died without blood being present in the gut. They died of renal failure from dehydration 7 to 10 days after admission. Is it necessary to give fluids only? Should one give a nutritious semi solid diet?

Both would eliminate these deaths from renal failure. Many clinicians favour a puree diet for it replenishes the depleted body proteins and re stocks the liver with glycogen. The patient is therefore in a stronger position should bleeding recur. There is no evidence that early feeding increases the risk of further hæmorrhage and clinically the patients do well.

Blood transfusion

The introduction of blood banks and the Medical Research Council standardisation of giving sets has greatly simplified the administration of blood. Nevertheless unpleasant reactions may still follow the most careful transfusion. It is therefore necessary to appreciate the exact purpose of the transfusion. It is very uncommon for a patient to bleed to death from the initial erosion of the blood vessel. In spite of a most profuse loss of blood and severe shock recovery is known to take place without transfusion. There is a risk that in some older patients prolonged shock may cause irreversible cellular changes so that death supervenes a few days later. But death is more often the result of the second, third, fourth or fifth hæmorrhage within the course of as many days. A blood transfusion after the initial hæmorrhage provides a reserve of blood should bleeding recur—it leaves him with a greater margin of blood to lose before the anoxæmic level is reached and puts him in a more favourable position should bleeding start again. There is no evidence that a *slow* drip blood transfusion increases the likelihood of further bleeding. If women have to be transfused during the reproductive period of life they should be tested for Rh agglutinins and receive Rh negative blood if they are Rh negative.

Surgery

The patients who are likely to die under medical treatment are those with known chronic ulcers whose bleeding recurs during the first few days. The mortality from medical treatment is about 50 per cent for those over 50 who have such *repeated* bleeding from a chronic gastric ulcer. Persistent ulcer pain after admission and thickened tortuous arteries are bad prognostic points. It is in this group that operation (partial gastrectomy) should be considered.

Occasionally a patient may be found to have pyloric stenosis with visible peristalsis. If this is the case it is advisable to

empty the stomach at night and arrange operation as soon as the general condition allows

MANAGEMENT OF GASTRO DUODENAL HÆMORRHAGE

- 1 Reassure the patient
- 2 Secure mental relaxation with morphine gr $\frac{1}{6}$ or injection of sodium phenobarbitone gr 3
- 3 Unless there is shock allow one to two pillows as desired
- 4 If there is shock raise foot of bed and give oxygen at 6 litres a minute by B L B mask
- 5 Order half hourly or hourly pulse chart
- 6 Take 5 c cm venous blood in an oxalate tube for hæmoglobin and blood urea estimation and 5 c cm in a plain tube for serum for Rhesus factor tests and direct matching in case of blood transfusion

Note —

- a A high initial hæmoglobin may be misleading as it takes at least six to eighteen hours for blood to be diluted after bleeding
- b A blood urea of 70 to 100 mgm per cent is common after a brisk hæmorrhage. If over 150 mgm per cent consider the possibility of dehydration chronic nephritis or alkalosis and give saline intravenously
- 7 Arrange for blood grouping of available relatives if there is no blood bank
- 8 Allow dilute saline (one part normal saline to two parts water) by mouth as desired in a feeding cup by the bed
- 9 Order semi solid diet *e g* —

6 a m	Cup of milky tea
8 a m	Porridge and Bemax or lightly boiled egg thin bread and butter and jelly marmalade cup of milky tea
10 a m	Cup of milk and biscuit
12 noon	Minced meat chicken or steamed fish mashed potato puree carrot or cauliflower
2 p m	Egg custard or cereal pudding or apple puree orange juice
4 p m	Cup of milky tea three slices of thin bread and butter bramble jelly sponge cake

6 p m Cream of vegetable soup or minced chicken sandwich

8 p m Milk pudding or cup of milk

10 p m Cup of milk and biscuit

Give milk feeds during the night if awake

If the patient cannot tolerate food (this is rarely so) or if there is pain or clinical evidence of pyloric stenosis give 7 fl oz milk feeds two hourly

- 10 If there is clinical evidence of a massive hæmorrhage and if the hæmoglobin is below 40 per cent give a blood transfusion at 40 drops a minute. It is rarely necessary to cut down on a vein

Note—The appearance of the patient immediately after a brisk hæmorrhage may be very misleading. The lips and face may be very pale from reactive vaso constriction. The colour and the initial fall of blood pressure may quickly improve. A review after 20-30 minutes may help in assessing the severity of the hæmorrhage. It is unwise to give the blood quickly.

- 11 Antacids may be given. Aluminium hydroxide $\frac{1}{2}$ oz two hourly is recommended as this has an anti peptic as well as an acid neutralising action and so may hinder the digestion of clot. Double doses at night may be given while awake. After three days it is sufficient to give $\frac{1}{2}$ oz four times a day or to revert to magnesium trisilicate or tribasic alkaline powder (equal parts of calcium and tribasic magnesium phosphate)
- 12 Give ascorbic acid 200 mgm three times a day for five days and then 50 mgm daily (or orange juice). Ascorbic acid deficiency retards healing of the ulcer.
- 13 Ensure sleep at night with chloral hydrate gr 20 well diluted or sodium amytal gr 3 or soluble barbitone (medinal) gr $7\frac{1}{2}$
- 14 The bowels are usually inactive for several days after a hæmorrhage. It is unwise to give aperients or early enemata. Reassure the patient and give a simple enema on the fourth day.
- 15 Give Ferrous Sulphate B.P. gr 3 three times a day preferably crushing the tablet before swallowing.

- 16 Arrange bed exercises for the patient Allow him up for a short time as soon as possible but preferably not until the hæmoglobin is 60 per cent and the stools negative for occult blood
- 17 Arrange a barium meal when the patient can get up
- 18 If an ulcer is found keep the patient under careful supervision until it is healed
- 19 Give instructions on suitable convalescent ulcer regime a certificate for extra milk and eggs and a note for his canteen to enable him to get a light mid day meal Arrange follow up of the patient

Note—However severe a hæmorrhage may seem and however unpromising the prognosis always persist with treatment Occasionally emptying the stomach and washing out with water may tip the scales in the patient's favour Oxygen at 6 litres a minute through a B L B mask and plasma or even intravenous saline may help greatly in the absence of blood For recurrent bleeding from duodenal ulcer a milk drip may be helpful

F AVERY JONES

CHAPTER VI

Medical Emergencies in Obstetrics and Gynaecology

IN this chapter, in addition to measures needed for the immediate emergency, treatment required subsequently is also mentioned since it must often be ordered at the time

OBSTETRICS

ABDOMINAL PAIN DURING PREGNANCY

Various types and degrees of abdominal pain are very frequent during pregnancy. The presence of the pregnant uterus may obscure common surgical causes of pain and make diagnosis difficult. Thus the possibility of an atypical appendicitis with the appendix tucked away behind the uterus or of intestinal obstruction as causes of pain and vomiting must be considered. Cholecystitis, too, is not uncommon during pregnancy. Pyrexia is not usually found with pain caused by the pregnant state itself nor is pain a frequent symptom of the vomiting of pregnancy.

EARLY PREGNANCY

The common causes of abdominal pain in early pregnancy are —

- Threatened abortion (*page 68*)
- Ectopic gestation
- Retroverted gravid uterus
- Stretching of the round ligaments
- Stretching of the uterus

Ectopic gestation.

This is mentioned because it is most important to differentiate it from medical causes of pain (and to treat it surgically)

The typical history is a short spell of amenorrhœa followed by sudden severe colicky pain in the lower abdomen usually felt unilaterally. Vaginal bleeding of a varying degree occurs. Frequently there is a history of 'one child sterility'. The

signs are a raised pulse rate with some degree of shock and hyperæsthesia of the lower abdominal wall with guarding rather than true rigidity. On vaginal examination there is exquisite tenderness in the pouch of Douglas and a tender mass may be felt there or in the lateral fornix.

In such a case the diagnosis may be very easy but there are others which do not conform to the typical picture. These are usually cases where the pregnancy has formed a mole which has not been extruded from the tube. In general the mass felt is very tender and if still in the tube is felt far out laterally towards the pelvic wall as distinct from the mass in salpingitis which is drawn down into the pouch of Douglas. If there has been any marked leakage of blood into the peritoneal cavity a useful accessory sign is the exquisite pain and tenderness elicited when the posterior fornix of the vagina is merely touched.

Retroverted gravid uterus

A retroverted gravid uterus causes lower abdominal pain and backache early in pregnancy. Treatment consists in correcting the position manually. If this is not possible the patient should be instructed to lie prone for 20 to 30 minutes at a time several times a day. The bowels should be kept open by mild aperients and the bladder emptied frequently. Intercourse should be forbidden. Usually the uterus rises into the abdomen and the pain is then cured.

Stretching of the round ligaments

Pain from this cause usually occurs about the twelfth to twentieth weeks of pregnancy. It is felt along the course of the ligament i.e. along a line from the mid point of the inguinal ligament to the side of the uterus and is more frequent on the right side. It is relieved by rest and no special treatment is needed.

Stretching of the uterus

This may cause indefinite pain at any stage of pregnancy. On abdominal examination the uterus is tender and the patient states that she feels tight there. These cases are difficult to explain as some occur in the early months before hydramnios is likely. Relief may be obtained by giving progesterone 10 mgm daily for a week or so. (In later months multiple pregnancy or

hydramnios may be responsible calling for prompt obstetrical treatment)

THE LATTER HALF OF PREGNANCY

The causes of abdominal pain at this stage of pregnancy are —

Breech presentation

Accidental hæmorrhage and placental separation

Degeneration of a fibroid

Pyelitis (*pages 39 and 235*)

Hæmatoma of the rectus muscle (*page 45*)

Breech presentation

This commonly causes pain in the fundus over the foetal head. The pain is characteristic of the malpresentation and can be relieved by version.

Accidental hæmorrhage

In this condition pain is usually over the placental site but there may be scattered areas of tenderness over the uterus caused by small hæmorrhages in its wall.

Treatment consists of rest, analgesics and vitamin E (Fertilol 1 capsule three times a day) in the less serious cases. Appropriate obstetric treatment will be indicated in others.

Degeneration of a fibroid

This may occur at any stage of pregnancy but is commoner in the latter half. The fibroid is felt as a tender discrete swelling. Most cases settle down with rest and analgesics.

ABORTION AS A MEDICAL EMERGENCY

The treatment of abortion is expectant until it is inevitable and when it is inevitable it is to see that it is complete. The emergency of abortion is therefore firstly medical but it becomes surgical if the products of conception are retained.

Patients threatening to abort are those known to be pregnant who have some vaginal loss without established pains or dilatation of the internal uterine os. Such patients should be put to bed at once and given a sedative such as omnopon gr $\frac{1}{2}$. This is preferable to morphine to which there may be idiosyncrasy. Progesterone 10 mgm is given intramuscularly and repeated

daily until the loss ceases or abortion occurs. Since oestrogens may help the action of progesterone synergically hexoestrol 5 mgm may be given three times a day. For patients who refuse injections one of the oral analogues such as ethisterone 10 mgm three times a day may be used.

In all such cases the uterine position should be ascertained. If retroversion is present the patient should lie prone for half hour periods frequently. The bladder should be emptied regularly and simple aperients used. Coitus should be forbidden till full placentation has taken place at four months.

Should abortion threaten at a rather later time than is usual *e.g.* at four or five months the abdominal pain and tenderness over the uterine fundus may lead to mistakes in diagnosis especially if the history of amenorrhœa is not obtained or is concealed. It should be remembered that degenerating fibroids are not common in young married women and that at this stage of pregnancy the uterus may be markedly cystic.

Pregnancy tests

It is sometimes important from the medico legal point of view to be able to establish whether at any given time a patient was pregnant or not. The value of the evidence often depends on the time when it was obtained and so its collection is a relatively urgent matter.

For the establishment of a diagnosis of pregnancy the biological tests (Asheim Zondek Friedman and Hogben) are of great value but must always be used in conjunction with the clinical findings. The A Z test is probably the most accurate but the Friedman test is easier to perform. In general the tests become positive within about a fortnight of the first missed period and become negative within a fortnight of delivery or abortion.

A positive A Z test indicates pregnancy in 98 per cent of cases (the exceptions being due to the menopause some tumours and minor degrees of thyroid imbalance) but a negative test does not carry such a high degree of accuracy. Negative Friedman tests may be wrong in 5 to 8 per cent of cases.

In making a diagnosis between secondary amenorrhœa and pregnancy it is important to bear these facts in mind since a clinical diagnosis may be wrongly negative also in early pregnancy because of the softness and flaccidity of the uterus.

Little help is gained by using pregnancy tests in cases of threatened abortion to determine whether or not the pregnancy is still continuing. Pregnancy tests depend on the production of hormones by the chorionic tissue. This may remain active and the Friedman test positive for 14 days or more after the foetus is dead. Similarly if the production of the hormone by the chorionic epithelium is deficient the test may be negative and lead one to believe wrongly that the pregnancy has come to grief. Should tests give results which seem at variance with clinical findings it is best to repeat them after waiting a fortnight. In general a negative or doubtfully positive test in a patient with a threatened abortion does not augur well for the continuation of the pregnancy as it points to possible abnormality of the chorionic tissue.

HYPEREMESIS GRAVIDARUM

Some degree of nausea or vomiting is conceded as a symptom of pregnancy. hyperemesis is a condition in which the degree of vomiting is so exaggerated as to distress the patient or affect her general condition. Faced as yet with an unknown ætiology treatment is mainly expectant prophylactic of possible complications (dehydration polyneuritis and liver damage) and observational of the patient's general condition to determine whether termination of the pregnancy may be necessary.

Treatment—In mild cases of hyperemesis the patient should be encouraged to continue her normal life reassured by the knowledge that the sickness is bound to diminish. The bowels should be made to act daily and for several days meat and all fatty foods should be avoided. Glucose and sugary foods such as jam and honey should be taken freely.

Should improvement not occur or if the vomiting is already affecting the general condition as indicated by a rise in pulse rate dehydration or acetonuria the patient should be put to bed in hospital away from the worries of her home. From the onset of treatment there careful records should be kept of the intake and output of fluids the pulse rate and temperature the presence and amount of acetone or albumin in the urine the blood pressure and the general clinical state. If vomiting is only moderate in amount fluids by mouth should be restricted to 2 pints for 24 hours and the diet should be of a carbohydrate

If emergency situations are to be avoided full co operation between the physician and the obstetrician is necessary as there is a danger of each assuming that the other is doing the supervising

Pregnancy does not appear to aggravate pulmonary tuberculosis or cause any specific emergencies but rapid deterioration often follows parturition. The sudden emptying of the uterus leads to increased respiratory movements and a rapid spread of disease may occur. To avoid this a pneumo peritoneum may be induced at once putting in 1 500 to 2 000 c cm of air (*page 383*)

Patients with pulmonary tuberculosis are often warned of the danger of pregnancy (but are not always instructed in how to avoid it). They therefore tend to become worried during pregnancy and the possibility of acute mental disturbance is always present

ANÆMIA AND PREGNANCY

Anæmia usually of hypochromic type frequently complicates pregnancy and may constitute an emergency if its degree is such that the patient's condition would be jeopardised by loss of blood at delivery. Such anæmia is usually seen in older multiparæ whose hæmoglobin levels have been progressively falling before pregnancy

If anæmia is diagnosed early in pregnancy treatment should be on the usual lines by massive iron or other therapy (*page 132*). If however it is only discovered late transfusion may be necessary great care being taken to avoid transfusion accidents by careful cross matching and the use of Rhesus negative blood

HYPERTHYROIDISM AND PREGNANCY

Hyperthyroidism is usually mild when pregnancy supervenes. Even so emergencies may arise. Thyroid crises should be dealt with as described on *page 218*. Myocardial failure is serious and justifies the termination of pregnancy

PREGNANCY IN THE DIABETIC

THE MOTHER —The emergencies which may arise are —

- (1) Ketosis particularly in early pregnancy because of the metabolic changes taking place
- (2) Hypoglycæmia may occur particularly towards the end of pregnancy and is attributed to lowering of the renal threshold

which allows excessive leakage of sugar from the blood to the urine. Probably it is the occasional *sudden* and transient fall of the renal threshold that accounts for symptoms such as fainting attacks of hypoglycæmic origin. In diabetes theoretical considerations do not always apply to individual cases and it is wise to be guided by the blood sugar levels.

With careful management both of these emergencies may be avoided and if they occur they should be treated on the lines laid down in chapter VIII.

(3) **Toxæmia.** There seems to be an increased risk of pregnancy toxæmia and the emergencies of eclampsia may have to be dealt with.

During labour the usual diet should be continued as long as the patient can take it and then 20 gm of glucose given every two hours with an extra 30 gm at the onset of the second stage. The risk of hypoglycæmia increases during labour because of muscular action and so insulin should be cut down until it is over. Half a unit should be given for each gramme of glucose retained.

THE BABY—Many of the emergencies confronting the baby are obstetrical in nature from the fact that he is often heavy and post mature. Hydramnios is frequently present also.

Neonatal death from hypoglycæmia due to over activity of the pancreas has occurred and the care of the diabetic's new born child should always have the priority of a potential medical emergency. Feeds of 10 per cent glucose should be given four hourly starting with 1 fl oz. These should continue until breast feeding is well established and if necessary up to a week. The state of the baby is the best guide. Occasionally a blood sugar estimation may be indicated.

Premature death of a foetus in utero has been thought to be more likely in those patients who develop hyperprolactinæmia. An attempt to avoid this risk can be made by suppressing anterior pituitary activity with large doses of oestrogens. A daily dosage of 25-40 mgm of stilboestrol by mouth or 150 000 units of oestradiol benzoate by intramuscular injection is needed from about the fifth to seventh months of pregnancy. This treatment is expensive and protracted. It would be best to confine its use to patients who have a history of foetal death in utero or in whom estimation of blood gonadotropes has been made.

PSYCHIATRIC DISORDERS AND PREGNANCY

(See page 194)

GYNAECOLOGY

Gynaecological emergencies usually present as abdominal pain, or with some other marked symptom such as menorrhagia. Accurate diagnosis is important as many patients will need surgical treatment either immediately, or when the emergency has been tided over. Conditions which justify medical treatment are the symptoms of menorrhagia, amenorrhœa, and pruritus the inflammatory lesions of salpingitis, and certain causes of abdominal pain in women, *e.g.* mittelschmerz.

MENORRHAGIA

Menorrhagia is merely a symptom, and therefore its cause must be sought in all cases. As this may be in the genital tract (fibroids or pelvic infection), vaginal examination must always be performed. It is often difficult to diagnose with certainty a small submucous fibroid causing menorrhagia as the uterus may only feel a little bulky and have no obvious deformity of outline. Exploration with a sound under anæsthetic may be necessary.

In many cases menorrhagia is caused by the gynaecological endocrine make up of the patient, or by some general condition such as hypothyroidism, leukæmia or anæmia. Minor degrees of alcoholism particularly if spirits are taken, may also cause excessive loss.

Menorrhagia due to essential functional causes may occur at puberty, during adult life (cyclic or acyclical bleeding) or at the menopause. Diagnosis is made by excluding general causes and local pelvic conditions by a consideration of the history and type of loss, and, if necessary, by endometrial biopsy.

Although most patients will complain of the excessive loss some, especially if the periods have always been heavy, will notice only the secondary effects of anæmia. Such cases tend to occur near middle life when the bone marrow response to hæmorrhage is waning.

Medical treatment may be necessary during the acute attack, to combat a known cause, to correct resulting anæmia, and to prepare a patient for surgery.

EMERGENCY SYMPTOMATIC MEASURES—The patient complains of a sudden drenching loss of blood and perhaps faints. She should be put to bed and examined to exclude any cause which calls for surgical treatment. Ergot administration is traditional and the following mixture should be ordered

Solution of Strychnine hydrochloride	m	3
Dilute hydrochloric acid	m	10
Liquid extract of ergot	m	30
Syrup of ginger	• m	30
Water to	fl oz	1

Three times a day after food

Alternatives are Ergotamine tartrate 1 mgm three times a day by mouth or injections of Pitocin 0.5 c cm every four to six hours

If the patient is a young girl (puberty menorrhagia) or if the history in an adult is suggestive of metropathia hæmorrhagica (*i.e.* spells or irregular excessive loss following short periods of amenorrhœa) help may be obtained from progesterone 10 mgm intramuscularly every day

When gross anæmia is present a blood transfusion should be carried out. In all cases it is essential to treat the anæmia by giving large doses of iron (ferrous sulphate B.P. gr. 3 as a pill or tablet four times daily after food)

Emergency treatment of the medical causes of menorrhagia—Thyroid gr. $\frac{1}{2}$ three times a day should be given if there is any evidence of hypothyroidism. Emergency treatment of blood diseases should be given if necessary (Chapter IX.)

Puberty menorrhagia is usually a self-limiting disease which corrects itself if given time. A high protein diet, vitamins, iron and calcium. If the loss is severe relief may be obtained by progesterone 10 mgm intramuscularly three times a week for the latter two weeks of the menstrual cycle.

In adults chorionic gonadotropic preparations are useful because of their presumed luteinising effect on the ovary. Doses up to 500 units daily during the second fortnight of the menstrual cycle may be necessary. If this fails methyl testosterone 5 mgm orally twice a day throughout the cycle frequently lessens the bleeding.

In patients nearing the menopause most endocrine preparations are of little help as the ovaries are growing insensitive to

stimuli Temporary inhibition of ovulation by testosterone propionate 25 mgm intramuscularly on alternate days may help these patients (It should not be continued for more than a few months because of the danger of masculinisation) More often an artificial menopause is indicated after careful exclusion of malignant disease

Emergency medical measures preparatory to operation—Patients whose hæmoglobin is less than 50 per cent are best tided over by medical treatment After acute blood loss transfusion may be necessary but is best reserved until just before operation In some cases the temporary use of testosterone will enable anæmia to be combatted

AMENORRHŒA

Amenorrhœa scarcely constitutes a medical emergency unless it is the presenting symptom of a serious underlying condition such as diabetes tuberculosis or pituitary tumour

The most common causes of amenorrhœa other than pregnancy are psychological or a change in environment Fear of an undesired pregnancy or worry that the menopause is starting early may in an unbalanced patient precipitate a serious anxiety neurosis In such a case relief may best be obtained by the induction of a 'period' Having excluded the possibility of pregnancy by careful examination prostigmine 1 c cm subcutaneously, for three consecutive days will usually cause a loss from uterine congestion

Secondary amenorrhœa may similarly cause and be part of the picture of an emergency situation As it has probably been present for some months or years no response to simple measures is likely and a course of œstrogen and progesterone therapy should be used Give 50 000 units œstradiol benzoate intramuscularly twice a week for two weeks followed by progesterone 5 mgm intramuscularly daily for five days A period will usually follow within a few days after the cessation of injections and the artificial cycle is repeated starting the œstradiol on the first day of the loss Gonadotrophic hormones may also be given if pituitary deficiency is thought to be present 500 units of follicle stimulating (serum) preparation are given every third day for five doses followed by 500 units of

a chorionic (urinary) preparation, *e g*, Antuitrin "S" daily for five days

Only a certain proportion of patients will respond to treatment, and it is necessary here to condemn the excessive and indiscriminate use of endocrine preparations

PRURITUS VULVÆ

Occasionally pruritus vulvæ is so marked that secondary psychological manifestations make its treatment that of an emergency. Diabetes must be excluded and any local vaginal, cervical, or rectal infection sought for and treated. In idiopathic cases it may be important to gain relief so that the vicious circle may be broken.

Adequate doses of a sedative, *e g* phenobarbitone should be given, and temporary anæsthesia of the vulval area achieved by the subcutaneous injection of proctocaine—a proprietary preparation of procaine and butyl *p* amino benzoate in almond oil.

Two small weals are raised in the labia majora with 2 per cent procaine the site chosen being midway between the clitoris anteriorly, and the anus posteriorly. Through these areas proctocaine is injected in a fanwise manner with a long needle, first directing it forwards towards the clitoris and leaving about 2.5 c cm of the solution along the course of the needle as it is slowly withdrawn. The needle is then swung round towards the anus and a further 2.5 c cm injected into the posterior half of the field. Thus, about 10 to 12 c cm in all are necessary. Usually relief can be obtained for a fortnight and this may break the circle or enable other treatment to be given. Proctocaine must not be used in the presence of infection.

EMERGENCY CONDITIONS OF INFLAMMATORY ORIGIN

Most cases of salpingitis, whether acute (due to the gonococcus or non specific organisms) or chronic (due to tubercle bacillus) are treated by non surgical methods unless there are definite reasons such as doubt in diagnosis or spreading peritonitis to indicate the need for surgical interference.

Diagnosis—The differentiation of unilateral salpingitis from right sided appendicitis is important and particularly if the appendix lies in the pelvis. Helpful points are—

- (a) The temperature—pulse ratio In salpingitis the pulse rate is proportional to the pyrexia whereas in appendicitis it is disproportionally rapid
- (b) Rovsing's sign Pressure is applied over the descending colon In appendicitis pain is felt in the right iliac fossa when the pressure is released
- (c) Headache is more frequently present in salpingitis than in appendicitis

Treatment—The patient is put to bed in Fowler's position and pain relieved by heat applied to the abdomen Compound tablets of codeine B P C two every four hours often suffice to relieve the pain but pethidine (100 mgm intramuscularly) or even morphine gr $\frac{1}{4}$ subcutaneously may be required Chemo therapy (by a sulphonamide or penicillin) should be used

Drainage may be aided by the vaginal application of tampons of ichthyol (5 to 10 per cent) in glycerin

The social and medico legal implications of salpingitis must be remembered If gonococcal the source of infection has to be traced and obviously the husband will have to be examined There is danger too of infecting children and this may make isolation important If salpingitis originates from an abortion the possibility of criminal interference must be considered (page 366)

VULVO VAGINITIS IN CHILDREN

Owing to its contagiousness this demands urgent treatment Swabs should be taken to identify the causative organism and the patient isolated All contacts should be examined Sulpha methazine should be given in dosages proportional to the age of the child the smallest infant receiving up to 3 grammes in the first 24 hours

Penicillin in cases of gonococcal origin is valuable 75 000 to 100 000 units being given intramuscularly during 24 hours Cases not of gonococcal origin do not respond so well to penicillin In view of the possibility of mixed infections masking the gonococcus it is probably wisest to give it in all cases

Apart from baths and subsequent thorough drying of the area local treatment is not usually necessary In resistant cases oestrogenic preparations may help (e.g stilboestrol 0.5 mgm twice daily)

TUBERCULOUS SALPINGITIS

This is always secondary to a tuberculous lesion elsewhere and therefore foci in the chest abdomen and urinary tract must be sought

If accompanied by ascites or areas of caseation in the omentum attacks of abdominal pain may occur. Diagnosis has to be made from other causes including ovarian cysts. Conservative medical treatment is best and consists of rest in bed particularly if there is pyrexia pain or a focus of infection elsewhere.

ACUTE LOWER ABDOMINAL PAIN IN WOMEN

Acute lower abdominal pain in women may have many medical causes as well as being caused by lesions of the genital organs themselves. The latter produce pain in various ways. The dull heavy ache of an ovarian cyst or fibroid is produced by its weight causing stretching of tissues or by congestion of its pedicle. The lancinating pain of a tubal mole is of the nature of colic. The premenstrual pain of endometriosis is due to tension. Peritoneal irritation causes pain from inflammatory lesions or extravasated blood and extension of malignant disease into nerves may cause severe pain.

Many patients need surgical treatment and only a few notes on diagnosis and medical treatment are here relevant. Ectopic gestation must be excluded in women of child bearing age who complain of marked unilateral pain and have a history of recent menstrual irregularity. In patients over 40 ill defined lower abdominal pain may well be a symptom of carcinoma of the ovary diverticulitis or carcinoma of the pelvic colon with pericolic abscess. The last two conditions present a tender mass in the left lateral and posterior fornices which may easily be confused with an atypical tubal or ovarian swelling if their possibility is not remembered.

Degeneration of a fibroid may cause acute pain and tenderness. Symptoms usually settle with rest and analgesics.

MITTELSCHMERZ

In younger women pain at the time of ovulation is not infrequent (Mittelschmerz). (See also page 40.) Whilst usually a dull ache it may be more acute if accompanied by free bleeding.

into the peritoneal cavity. In such cases the diagnosis of appendicitis is often made and an unnecessary appendicectomy performed. The timing of the attack is typical (twelfth to fifteenth days of the menstrual cycle). There is often marked hyperæsthesia on moving the cervix, and in the Pouch of Douglas, from accumulation of blood there. A leucocyte count fails to reveal the leucocytosis so typical of a suppurative lesion. Treatment consists of rest in bed and the administration of analgesics. Operation is needed only if the diagnosis is in doubt, or if marked intra peritoneal bleeding is present.

PAIN IN MALIGNANT DISEASE

Advanced malignant disease may cause urgent pain from nerve involvement. Intrathecal injection of absolute alcohol often gives marked relief, but in view of the dangers this method should be used only in this type of case.

The technique is as follows. The patient lies with the painful side uppermost. The pelvis is raised by a pad so that the sacral and lumbar regions are elevated. The head is lowered and the body turned ventrally so that the posterior sensory nerve roots are uppermost. The skin is cleaned and thecal puncture performed in the fourth lumbar interspace. When C S F is seen to escape, 0.5 c.c. of absolute alcohol is injected very slowly, drop by drop, taking two minutes to do this. No attempt is made to mix alcohol with C S F in the syringe. The patient is kept on her side for two hours. The patient will complain of numbness which disappears spontaneously.

KENNETH BOWES

CHAPTER VII

Respiratory Emergencies

THE chief conditions of the respiratory system which are likely to call for urgent measures are those in which respiration ceases or becomes painful or difficult or in which cyanosis occurs or blood is coughed up

Cyanosis and dyspnoea are commonly associated in respiratory emergencies and as a clear understanding of their mechanism is important in assessing their significance in an emergency they will be considered first

CYANOSIS

Cyanosis or blueness of the skin and mucous membranes is rarely in itself responsible for an urgent call but rather the associated symptoms. Its appearance depends on the amount of reduced hæmoglobin in the blood. If this is 5 gm per 100 c cm or more cyanosis appears. It may occur independently of respiratory failure as in some cases of congenital heart disease when venous blood crosses in the heart to the arterial side. In polycythæmia cyanosis occurs because the total hæmoglobin is so high that even with perfect pulmonary ventilation there are always at least 5 grams per cent of reduced hæmoglobin. Conversely in severe anæmia with hæmoglobin less than 5 grams per cent cyanosis is impossible.

The explanation of cyanosis in pneumonia and other lung diseases often presents difficulty. It does not follow that cyanosis will develop because a whole lung is out of action as in complete collapse the collapsed lung is nourished via the bronchial arteries and the blood flow through it from the pulmonary artery to vein is practically abolished. Thus there is no unoxygenated blood flowing back to the left auricle and therefore no cyanosis.

In lobar pneumonia cyanosis occurs because there is some blood flow through the solid lung. But oxygen will not alleviate it because oxygen cannot reach the blood via the solid lung and the blood flowing through the healthy lung is completely saturated with oxygen.

into the mouth. Two of the silk threads are attached to it and as it is withdrawn the plug impacts in the choana. The nose is then packed with oiled gauze. One piece of silk passes through the mouth and is attached by adhesive plaster or a bandage to the face for use in removal of the plug.

HÆMOPTYSIS

The spitting of blood tinged sputum or streaking usually calls only for reassurance as an emergency measure. In serious hæmorrhage from the lower respiratory tract blood wells up into the mouth without a cough. This is true hæmoptysis—the result of broncho pulmonary hæmorrhage.

Sometimes the patient is uncertain whether the blood was coughed or vomited and indeed some expectorated blood may be swallowed as during sleep and vomited later. Vomited blood is usually dark and may be acid (use *glad* litmus paper to test it) whereas coughed blood is bright frothy mixed with sputum and not acid.

It has been said that a patient never dies of hæmoptysis but after it from the spread of tuberculosis caused by retention of infected blood clot. This is not strictly true even of tuberculosis but it emphasises the danger of failing to cough up the blood. When hæmoptysis is of large amount it is either profuse and rapidly fatal as from a ruptured aneurysm or is but a small part of the clinical picture of some other condition.

Treatment—Only enough morphine should be given to allay anxiety (say gr $\frac{1}{8}$) but the cough reflex must not be abolished. Since in the rare cases of fatal hæmoptysis death occurs within a few minutes it is well to reassure the patient who has survived this period that he is not about to die. This is an important part of the treatment. An ice bag may be applied to the chest for its psychological effect.

If the probable side of bleeding is known the patient should be encouraged to lie on it. Screening has shown that this position does not diminish movement of the lower side but rather increases diaphragmatic movement there. This is unimportant and the real reason for lying on the side from which blood is coming is that it lessens the chance of blood entering the bronchi of the opposite side. Flat sandbags on the affected side may

help to lessen the movement until the patient becomes too tired to support their weight

One of the following measures which are said to favour blood clotting may be used. None of them is very effective

- 1 5 to 10 c cm of 1 per cent congo red intravenously. A glass interceptor between the needle and syringe enables one to be sure by seeing blood in it when the piston is withdrawn that the vein is entered
- 2 5 c cm of 1 per cent sodium chloride intravenously
- 3 Half a teaspoonful of common salt by mouth
- 4 10 c cm of 20 per cent calcium gluconate intravenously given slowly
- 5 Coagulen Ciba 5 c cm intramuscularly
- 6 Blood transfusion. A small direct arm to arm transfusion is best (*page 435*)

It has been recommended that if the patient is known to have tuberculosis with unilateral cavitation or if a radiograph demonstrates this a large pneumothorax (up to 1 000 c cm) may be induced. This has been done successfully but in my opinion is hazardous and may be ineffective because of adhesions. A simpler and equally effective procedure is to induce a pneumoperitoneum (*page 383*). Since the resulting rise of the diaphragm is bilateral the affected side need not be known. If it is known collapse can be increased by a phrenic nerve crush on the side of the cavity.

ASTHMA

When called suddenly to a previously unknown patient in a paroxysm of supposed asthma it is well to remember Chevalier Jackson's famous aphorism—All is not asthma that wheezes.

Asthma with unilateral signs suggests bronchial obstruction. The sudden appearance of asthma in a middle aged hypertensive subject suggests left ventricular failure (cardiac asthma). In such a patient moist sounds in the lungs are a marked feature in contrast to the dry sibilant rhonchi of true asthma.

When real doubt arises as to the pulmonary or cardiac origin of the dyspnoea an estimation of the circulation time will help. 2.4 grams of saccharine dissolved in 4 c cm of distilled water are injected into a cubital vein. The time taken until the patient tastes saccharine is noted (arm to tongue time). It is normal

(up to 17 seconds) in asthma, but prolonged in left ventricular failure because of its slower passage through the congested pulmonary circulation

Treatment—Solution of adrenaline hydrochloride B P 1 in 1,000 should be injected subcutaneously at once. The earlier it is given the more likely it is to be effective. Up to 5 minims may be enough, but if wheezing persists this dose may be repeated every few minutes. It is important to make sure by withdrawing the piston, that the needle point is not in a vein. Intravenous injections of adrenaline cause faintness, pallor, palpitation and collapse.

In 'status asthmaticus' a large dose (up to 120 m) may be needed. The needle should be left in situ strapped with the syringe to the skin, and 1 minim injected every 30 to 60 seconds. Alternatively, one of the slowly acting preparations may be used to keep up the effect. They are—

- 1 Adrenaline in oil (Parke Davis & Co) 1 c cm contains 2 mgm of adrenaline hydrochloride (= m 30 of solution of adrenaline hydrochloride B P)
- 2 "Hyperdure" adrenaline (Allen & Hanbury) This is adrenaline with mucic acid, dose 3 to 8 minims (0.18 to 0.5 c cm)
- 3 Adrenutol (Evans) This is a solution of adrenaline 1 in 500 and chlorbutol 1 in 500 in water and glycerine (dose, 1 c cm)

When a patient fails to respond satisfactorily to adrenaline a useful remedy is aminophylline 0.25 gm in 10 c cm intravenously, or 0.5 gm in 2 c cm intramuscularly. It is thought to sensitise the patient to adrenaline. The dose may be repeated in four to six hours.

For intractable cases of "status asthmaticus" intravenous paraldehyde is recommended. It may be taken straight from the bottle into the syringe and given slowly into a vein in doses of 3 to 4 c cm for an adult, or it may be diluted 10 times with warm (98° F) normal saline and given by slow intravenous infusion until the condition is brought under control.

Adrenaline 1 in 100 by nasal spray, or one of the proprietary preparations containing it may be used. A convenient apparatus for the latter is the Collison Inhaler, which is operated by compressed air or oxygen. It may be hired from the Inhalation

Institute 87 Eccleston Square London S W 1 (Telephone VICTORIA 1676) Telegrams Idac London

Inhalations of adrenaline solutions stronger than 1 in 1 000 should be used with caution and only a few puffs of vapour inhaled. Otherwise adrenaline poisoning shown by pallor anxiety tremor and tachycardia may occur.

Individual cases may test the physician's ingenuity to the utmost. It must not be forgotten that some other condition such as pneumonia may underlie and be masked by the status asthmaticus. I have known attacks to be complicated by recurrent collapse of the lung resulting from bronchial obstruction by thick mucus.

It is tempting to use morphine but this will nearly always be regretted particularly in the elderly and those who cough up plugs of mucus. If it has to be used it should be combined with atropine gr $\frac{1}{64}$ to lessen bronchial secretion and relax bronchial muscle. It is best reserved to ease the passing of the rare moribund case of status asthmaticus. In a prolonged attack which is wearing the patient out it is much wiser to use sodium phenobarbitone gr 3 intramuscularly or paraldehyde as indicated above.

Occasionally marked tachycardia will preclude the use of adrenaline. In such cases posterior pituitary extract 0.3 to 0.5 c cm may be effective but it is dangerous in elderly subjects who may have coronary ischaemia. Posterior pituitary extract may be combined with adrenaline (Kadamysin 1 c cm formerly called Asthmolysin Chas Zimmermann & Co Ltd).

An electric fan placed before the patient will help to make him more comfortable especially if the atmosphere is warm and humid. Sometimes a prolonged attack which does not respond to the above measures will be relieved by the aspiration of mucus through a bronchoscope. A mixture of oxygen 20 per cent in helium administered at a pressure of 4 c cm of water brings prompt relief but this treatment is not readily available in Great Britain. Occasionally the treatment of asthma will be part of the treatment of aspirin poisoning (*page 8*).

PLEURAL PAIN

Pleural pain whether due to pleural tension or pleural friction has the same characteristics. It is sharp and knife like

worse on inspiration or coughing, most marked over the bases of the lungs and relieved by pressure over its site

It may be associated with the onset of inflammatory or embolic changes in the lung, with pneumothorax and, very occasionally, with fracture of a rib from coughing. The treatment of the symptom is —

- 1 To allay pain and cough by morphine or codeine. Spraying the skin over the painful area with ethyl chloride until blanching occurs will sometimes bring relief
- 2 To immobilise the chest by strapping (which must go beyond the middle line front and back). This is not well tolerated by older patients with rigid chests. For them, a poultice and bandage is preferable

INSTRUCTIONS FOR STRAPPING THE CHEST FOR PLEURAL PAIN —

Eight pieces of strapping, two inches wide, and six inches longer than the hemithorax, are required. Sit the patient up and face his good side. Apply the centre of a piece of strapping to the mid axillary region of the painful side. Hold one end of the strapping in each hand. Place the knee against the good side of the chest (or have an assistant press against this side). Ask the patient to hold his breath in full expiration, and while he does so, attach the plaster to the skin round the chest. Repeat the procedure until the eight strips are used, overlapping them a little.

A small artificial pneumothorax (250 c cm) may relieve the pain if friction be its cause, but is only advisable if pain is persistent and other measures have failed.

MASSIVE COLLAPSE OF THE LUNG

This is a form of absorption collapse which comes on comparatively suddenly as a result of an obstruction of a large bronchus by viscid secretion and possibly from other causes not fully understood. It is usually a post operative emergency and not always very sharply marked off from, what is probably a rarer condition true post operative inhalation pneumonia. It has been shown to occur most often after upper abdominal operations and is more frequent after local, rather than general, anaesthesia because of the heavier premedication with morphine when local anaesthesia is used. Morphine, by depressing the cough reflex,

and atropine by rendering bronchial secretion scanty and viscid may play a part

About 24 hours after operation the patient suddenly becomes dyspnoëic and cyanosed and has discomfort in the chest which rarely amounts to pain. The temperature rises to 101° F and there is a short dry cough ineffective because of the pain it causes in the wound.

The most important single means of diagnosis is a radiograph of the chest and this should be made in any patient who soon after operation shows evidence of respiratory trouble. It shows a uniform ground glass opacity extending outwards from the hilum and often clearing towards the periphery. When a large area of lung is involved the heart will be seen to be moved over to the affected side and the diaphragm to be raised on the affected side.

In the absence of an X ray picture reliance must be placed on physical signs. These are restricted movement and impaired resonance and air entry together with evidence of mediastinal displacement to the affected side. The latter is hard to detect unless the collapse is massive. It is absent when collapse is bilateral. Moist sounds are rarely present.

Treatment—The object of treatment is to remove the bronchial obstruction. This may be done by bronchoscopy. As this may not be readily achieved an equally good and often more rapid result can be obtained by the manœuvre discovered by Sante in 1927.

The patient lies on his back and is then gently rolled back and forth about a dozen times the affected side being uppermost. A dose of cough mixture containing ammonium carbonate and anti spasmodics such as—

Ephedrine hydrochloride	gr $\frac{1}{4}$
Ammonium carbonate	gr 10
Sodium iodide	gr 5
Tincture of lobelia	℥ 5
Chloroform water to	$\frac{1}{2}$ fl oz

is given and the obstructing material may then be coughed up. Coughing can also be induced by the intravenous injection of 1 c cm of nikethamide. The manœuvre may be repeated four hourly and helped by springing of the ribs and heavy percussion of the affected side. This may seem a formidable procedure for

a patient with an upper abdominal wound but the condition is a serious emergency and warrants vigorous treatment. Lest incomplete aeration should lead to pneumonia it is wise to commence chemotherapy.

Oxygen may be given but CO_2 is contra-indicated as the hyper-ventilation it causes may lead to aspiration of material into the smaller bronchi. In desperate cases with great pain and dyspnoea and marked mediastinal shift due to high negative intrapleural pressure an artificial pneumothorax sufficient to restore the intrapleural pressure to normal will give marked relief.

PULMONARY EMBOLISM

Pulmonary emboli are of two kinds—(a) small and often multiple (b) large and single. Thrombotic pulmonary emboli originate in the peripheral veins when they are the seat of phlebitis (though rarely from varicose veins) or from post-operative thrombi in the pelvic and abdominal veins. They may also come from the right side of the heart in bacterial endocarditis and from mural thrombi in the right auricle in auricular fibrillation or in the right ventricle following a cardiac infarct.

Thrombosis follows embolism and a pulmonary infarct develops after a short interval. In some cases of congestive cardiac failure pulmonary thrombosis without previous embolism is responsible. The symptoms produced by a small embolus are pleural pain followed by hæmoptysis. In less sudden cases rise of temperature and pulse rate may be observed first. Sometimes there is only tachycardia and a sense of oppression in the chest. Physical examination reveals either nothing at all or perhaps a friction rub. X-ray examination may show an infarct but with small emboli this evidence may be absent.

Massive pulmonary embolism produces a cardiovascular emergency of the first order. (See also page 126.) The patient—usually the subject of phlebitis particularly of the deep leg veins or of a recent pelvic or abdominal operation—is suddenly seized with urgent dyspnoea and may have marked substernal pain. He is shocked, cyanosed and very restless indeed so that he throws himself about in the bed. A typical symptom is an urgent desire to defæcate. Only when 60 per cent of the pulmonary artery lumen is occluded does the cardiac output fall.

and so a large elongated thrombus in the pulmonary artery may be symptomless until it suddenly twists on itself and occludes the lumen. Death is fairly sudden though never so quick as in thrombosis of a main coronary artery. Many post operative cases have occurred at the tenth day when the platelet rise is at its height and the patient begins to move more freely in bed.

On examination the heart sounds are feeble. Sometimes there is auricular fibrillation and a loud pulmonary systolic murmur. Air entry is normal at all areas, and there are no râles. An E C G shows various changes such as depression of the S T interval in lead II—but none of these is diagnostic.

When pulmonary embolism is not rapidly fatal the condition of acute cor pulmonale described by Paul White may develop. It arises from dilatation of the right heart following increased pressure in the pulmonary circuit. It is characterised by pulsation in the second and third left interspaces and a friction rub there, dilated pulsating jugular veins, triple rhythm, fever, leucocytosis and the signs of a pulmonary infarct. The condition may subside in a few hours or last a few days until death occurs.

Diagnosis—The other acute chest calamity with which pulmonary embolism may be confused is coronary thrombosis. A history of anginal attacks, a marked fall of blood pressure and the absence of phlebitis are points in favour of coronary thrombosis. Marked cyanosis, dyspnoea and restlessness are in favour of pulmonary embolism especially when there is a possible source of an embolus. The pain of pulmonary embolism rarely spreads to the arms as does that of angina. Blood stained sputum favours embolism.

Treatment—Small pulmonary emboli are warnings of a more severe catastrophe. Thrombosis of leg veins from which such emboli mostly originate is a fairly silent process and so should be looked for carefully and local signs such as that of Homan's sign (calf pain on dorsiflexing the foot with the knee straight). If found the physician should not neglect the help of his surgical colleagues. Ligation of the femoral vein or even the inferior vena cava may be needed. Anticoagulant therapy by heparin and dicoumarin (page 439) should be used for several days with a view to preventing further clot formation. Heparin, which is effective at once, should be discontinued after forty eight hours by which time dicoumarin will be acting.

When massive pulmonary embolism occurs oxygen and nikitamide should be given. It has been suggested that part of the clinical picture results from reflex coronary spasm and for this reason intravenous injection of papaverine gr 1 to 2 with atropine gr $\frac{1}{16}$ is recommended.

Since pulmonary embolism does not always kill a patient in a matter of minutes there is a possibility of surgical treatment if one is prepared for it. In 1938 I witnessed the first successful pulmonary embolectomy in England (and the twelfth in the world). A woman aged 49 had developed phlebitis of the left internal saphenous vein following prepatellar bursitis. 10 days after admission she had chest pain and blood stained sputum but not until 25 days later did she suddenly become blue and dyspnoeic. She was taken to a specially prepared theatre where Mr Ivor Lewis opened the chest under local anaesthesia and after waiting until she was in articulo mortis incised the pulmonary artery and extracted a clot 16 inches (40 cm) long one hour and 20 minutes after the onset of acute symptoms. Circulation had been absent for three minutes and respiration for six minutes. After an amazingly eventful convalescence including a second smaller embolus she recovered.

Such heroic operations will not often be practicable and a simpler procedure would be to give heparin at once in the hope that by preventing addition to the embolus by thrombosis sufficient circulation will be maintained to save life.

FAT EMBOLISM

This is considered here because its main manifestations are pulmonary. A few days after a bone injury dyspnoea cyanosis and mild pyrexia appear. Examination reveals scattered fine rales and an X ray film may show diffuse hazy shadow. Confirmatory evidence is provided by a petechial rash and fat globules in the urine. Similar chest symptoms have followed calpiningography by iodised oil either immediately or several days later. In such cases X ray films of the chest have shown dense linear basal opacities caused by opaque oil in the smaller pulmonary arteries. When fat reaches the brain delirium and coma result but localising signs are absent. An emergency call may be occasioned by a sudden worsening of the patient's condition or by the rash or the cerebral symptoms.

Treatment is symptomatic by the administration of oxygen and nikethamide 10 c cm of 20 per cent sodium desoxycholate intravenously every two hours has been recommended

SPONTANEOUS PNEUMOTHORAX

When this causes urgent symptoms it is usually large and complete. Exertion may or may not determine its onset and the patient is often a young healthy person. There is sudden sharp pleural pain followed as the lung collapses by dyspnoea.

The classical signs are lack of movement on the affected side with resonance and auscultatory silence together with displacement of the apex beat to the opposite side. If the pneumothorax is small pleural pain alone may constitute the emergency. In such a case the pneumothorax will only be diagnosed accurately by X ray examination. The urgency of symptoms arising from a pneumothorax depends mainly on —

- 1 the tension within it
- 2 the degree of mediastinal displacement
- 3 the presence of bleeding (*see Tension pneumothorax page 103*)
- 4 the severity of the pleural pain

Although symptoms are most marked when intra pleural pressure is high they may be urgent also when the pressure is negative if the patient's vital capacity is diminished by emphysema.

TENSION PNEUMOTHORAX — This pneumothorax suffocans is the most serious type. It renders one side of the chest immobile and bulging and constitutes a real threat to life because by pressure on the great veins it interferes with the return of blood to the heart. It is detected by —

- 1 Displacement of the apex beat to the opposite side
- 2 Increasing cyanosis and dyspnoea
- 3 Increasing rapidity of the pulse
- 4 Excessively high positive pressure

Downward displacement of the liver by a right sided tension pneumothorax is a late sign as is also the change in the percussion note from resonant to dull when the tension is great. Such evidence should not be awaited.

The mechanism of tension pneumothorax is disputed. Pneumothorax following needling of the chest is usually simple but very rarely a tension pneumothorax may develop. In such

a case it seems possible that the hole in the lung may open with inspiratory expansion of the lung and close on expiration much in the same way that a tyre puncture becomes more obvious on inflation

Another mechanism which applies particularly to tension pneumothorax following injury is that an alveolus ruptures and causes interstitial emphysema of the lung and later of the mediastinum. The mediastinal air subsequently ruptures into the pleural space. Tension in the pneumothorax is built up during the forced expiration of coughing. This mechanism will explain pneumothorax which appears on the side of the chest opposite to the injury. The presence of subcutaneous emphysema of the neck in these cases supports this theory.

A third and most likely mechanism is the same as that causing a tension cavity, namely a valve like action of the bronchus. When the bronchus leading to the rupture on the lung surface is kinked during expiration or partially obstructed by caseous bronchitis or bronchial spasm it may allow air to pass on inspiration but not on expiration and so enable a positive pressure in the pleural space to be built up.

Treatment—Morphine, brandy and oxygen are the first things to use. If respiratory distress persists a needle should be inserted in the fifth or sixth space in the mid axillary line and connected with a manometer. If the pressure is positive air should be aspirated by a reversed pneumothorax apparatus. In extreme urgency a needle by itself will release air and save the patient's life. Should urgent symptoms recur it is best to leave the needle in the chest and connect it to a rubber tube which dips under water. The pressure at which the air blows off can be adjusted by altering the depth to which the tube is immersed. In this way air can be expelled via the needle but cannot enter it. This is insufficient in some cases and continuous suction may be needed.

This may be arranged by connecting the needle to a Sprengel's pump with a water bottle intervening to indicate if air is being aspirated. A manometer should be connected to a side arm of the needle. If there is no nearby water tap suction may be arranged by blowing oxygen through a filter pump, the jet of which is very fine. This may conveniently be fastened to a board such as that which holds the manometer of an A.P.T. apparatus.

(Fig 11) It is wise to protect the outlet of the pump by wire gauze. A flow of 4 litres per minute will produce a negative pressure of 8 c cm of water.

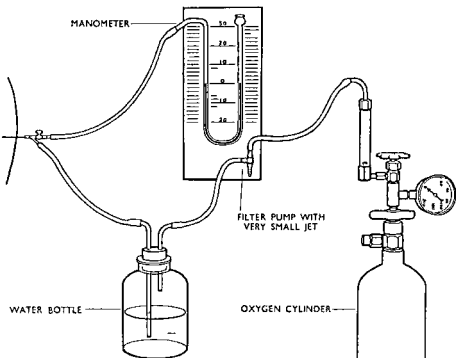


FIG 11

Diagram of apparatus for continuous suction of pneumothorax



FIG 12

Reversed non return valve from Army blood transfusion set

If a patient with a tension pneumothorax has to be moved as after an accident a useful device suggested by Major H Fuld is the non return valve from an Army blood transfusion set. This normally lets air into the blood bottle and prevents its escape so it must be attached to the needle in the chest in the reversed position (Fig 12).

Even when air has ceased to escape it is not safe to leave the patient for symptoms may recur and fluid particularly blood may accumulate

SPONTANEOUS HÆMO PNEUMOTHORAX

In every case of spontaneous pneumothorax the physician should bear in mind the possibility that blood as well as air may be escaping into the pleural cavity. The blood comes from the parietal end of the adhesion torn by the collapsing lung.

After the sudden onset of pneumothorax there is a period of temporary improvement lasting some hours or even days and then increasing pallor increasing rapidity of pulse dyspnoea and restlessness indicating internal hæmorrhage. Physical signs of air and fluid in the pleural cavity will be found. Abdominal symptoms resembling a surgical emergency are more apt to occur in hæmo-pneumothorax than in pneumothorax because of irritation of the diaphragmatic pleura by blood. It is wise therefore to put a patient with a spontaneous pneumothorax on a half hourly pulse chart.

A surprising amount of blood may be found in the pleural cavity even when the bleeding point is small. This is because of the suction of the negative intra pleural pressure and the fact that movement prevents clotting by defibrinating the blood.

Treatment—The patient should be treated as for spontaneous pneumothorax and transfused if necessary. Aspiration of blood should not be delayed longer than 24 hours lest fibrin deposit interfere with expansion of the lung later.

When it is clear that bleeding continues the blood should be removed through a large thoracoscopy cannula and an attempt made to cauterise the bleeding point. Only rarely however can this be seen through a thoracoscope and it may be necessary to ask a surgeon to open the chest.

MEDIASTINAL EMPHYSEMA

This results from rupture of pulmonary alveoli giving rise firstly to interstitial emphysema of the lung and later as the air spreads to mediastinal emphysema. Numerous conditions may cause this rupture such as trauma including artificial pneumothorax therapy and blast injuries and all the causes of increased intrapulmonary pressure such as cough and straining.

with the glottis closed. Sometimes an alveolus appears to have ruptured spontaneously in the absence of any known cause of increased pressure.

If of sufficient amount mediastinal air may escape —

- 1 Into the neck (Subcutaneous emphysema)
- 2 Into the retro peritoneal tissues
- 3 Into the pleural cavities (Spontaneous pneumothorax)

Diagnosis — Small amounts of air in the mediastinum are symptomless. Large amounts cause pain and retrosternal oppression like that of coronary disease and also dysphagia. Diagnosis may be difficult unless subcutaneous emphysema occurs. This by lowering the tension alleviates the pain. Other important signs are obliteration of the cardiac dullness and peculiar crackling and bubbling sounds synchronous with the heart beat and accentuated in systole (pericardial knock or Hamman's sign). Pericarditis and pneumopericardium are mimicked.

More urgent symptoms are caused by pressure on the big veins so that return of blood to the heart is impeded. Dyspnoea, cyanosis and pulmonary oedema result and death may occur from circulatory standstill. Convulsions from cerebral congestion are possible. These symptoms are the result of air block in which there are two elements: obstruction to the return of venous blood to the heart and interference with respiratory movements of the lung. I have seen the escaped subcutaneous air constitute an emergency because the patient's face and neck became rapidly distended causing respiratory obstruction.

Treatment — This becomes urgent when pressure symptoms appear. Probably the most effective measure is to incise the tissues at the root of the neck, introduce a catheter and apply suction. Tracheotomy instruments should be prepared. If unilateral lung disease is present it is reasonable to assume that the air leak is on the diseased side and to try the effect of collapsing this lung by artificial pneumothorax. On the only occasion in which I had to deal with this emergency this method was successful.

PNEUMONIA AS A MEDICAL EMERGENCY

In these paragraphs acute lobar and acute lobular or broncho pneumonia are not differentiated. All acute types of pneumonia are considered together and may be equally urgent except virus

pneumonia (primary atypical pneumonia) This is a less serious disease and unlikely to present an emergency (For pneumonia in children see page 256)

In all patients over 40 years of age the onset of pneumonia should be regarded as an emergency In younger patients the urgency depends on the severity of the attack and the previous state of the patient's essential organs

Pneumonia is by nature a local disease of the lung which strives to become general and in so far as the evidence of bacteraemia and involvement of other organs is greater so the urgency of the condition increases Pneumonia may cause an emergency situation —

- 1 By presenting as a grave undiagnosed pyrexial illness
- 2 Because of some urgent symptom arising in a patient known to have pneumonia

Diagnosis — This may be difficult in the early stages In a typical case the onset is sudden with shivering and rigor a high temperature a quick bounding pulse and a hot dry skin Evidence pointing to trouble in the chest such as rapid breathing pleural pain and a hard cough with viscid rusty sputum makes the diagnosis easier Dulness on percussion and noisy breathing confirm this but it must be remembered that signs may be atypical because the consolidation is deep or the bronchus blocked In central pneumonia diminished movement on the affected side is an important sign

Management — There are certain urgent features in the general management of the case If it seems doubtful whether a very ill patient can be nursed at home removal to hospital should be arranged early while there is still hope rather than later as a result of despair and defeat Should home be decided upon choose early the best room This may be downstairs to save wear and tear on the legs of relatives (and the doctor) Pneumonia occurring at awkward times such as holidays calls for foresight in ordering essential drugs and oxygen

It is wise to be forearmed about prognosis by knowing the causal organism Sputum must be saved early in the attack so that the organism may be identified and kept for tests of sulphonamide and penicillin sensitivity if these become necessary

Treatment — The choice of drugs facing the doctor is between penicillin and a sulphonamide In all cases where the use

of sulphonamides might be risky penicillin should be used. Such cases are those with anæmia leucopenia and conditions likely to hinder urinary excretion of sulphonamide (nephritis cirrhosis and cardiac failure). In severe cases particularly over the age of 40 both drugs should be given.

Penicillin is given in doses of at least 100 000 units in 24 hours (*see* *page* 442). Sulphamezathine is the sulphonamide of choice because it does not easily crystallise out in the urinary tract. (For dosage *see* *page* 440.) A mixture of sulphonamides (Sulphatriad May & Baker) has the same advantage (*page* 442). When the intravenous route has been impossible and swallowing difficult I have given sulphonamides through a stiff stomach tube.

Oxygen should be given early. In the first few days sleep at any price is a justifiable maxim and morphine should not be withheld.

Urgent symptoms and complications

Many urgent symptoms are discussed elsewhere: pleural pain (*page* 96), acute abdominal symptoms (*pages* 44, 112, 120), mediastinal emphysema (*page* 105) and acute mental symptoms (*page* 190). Pneumonia may be a complication of other emergencies such as diabetic coma, barbiturate poisoning and cerebral vascular accidents. Jaundice is a rare but serious complication and should be treated as described under hepatic failure (*page* 57).

Abdominal distension or meteorism is serious because it interferes with the action of the diaphragm. The rectum should be examined and if full of faeces an enema given. A flatus tube should then be passed and retained while drugs are used. Carbachol—a parasympathetic stimulant—gr $\frac{1}{10}$ to $\frac{1}{40}$ is given by subcutaneous injection. (It must not be given intravenously.) If this fails posterior pituitary extract 0.5 c.c.m. and eserine salicylate gr $\frac{1}{10}$ subcutaneously may be tried.

While the urgent nature of the illness may be obvious from delirium, severe pain and other symptoms, these may be absent even in a severe case. Because of pre-existing mental disorder a patient may show little reaction to pneumonia. Alternatively a previously normal brain may be so poisoned that the patient fails to show distress and remains calm and comfortable. Euphoria in pneumonia may therefore be an ominous signal. As an old aphorism warns us—Beware of pneumonia that smiles at you.

Overwhelming infection is shown by evidence of pericarditis and meningitis. The latter should be looked for especially since if penicillin is going to be used it is ineffective unless given intrathecally. I have seen pneumococcal pneumonia respond to penicillin while unrecognised meningitis progressed.

Medical shock in pneumonia (See also page 116)

This is usually described by the less arresting term peripheral circulatory failure. It arises essentially from disproportion between the capacity of the vascular bed and the volume of the blood in it.

In traumatic shock, diabetic coma and severe diarrhoea the circulation may fail because the circulating blood volume falls. In the shock of acute infections capillary dilatation from paralysis due to bacterial toxins is thought to be the mechanism.

The state of the peripheral circulation may be gauged clinically by observing the pulse, the arterial blood pressure, the venous blood pressure (as shown by the height above the heart at which the hand veins collapse) and by looking for capillary pulsation (as shown by gentle pressure on the finger nail or by noting rhythmic dimming of the illuminated finger pulp).

In medical shock the pulse is rapid and thready, the arterial and venous pressures are low, capillary pulsation may be present, the breathing is shallow and the skin cold and moist. This picture complicating pneumonia was formerly ascribed to cardiac failure. Although toxæmia and anoxæmia may damage the heart muscle it is peripheral and not central circulatory failure which is the main factor. The venous return is inadequate to maintain the circulation. The well runs dry.

Treatment—Warmth is important. The blood supply to the brain may be improved by raising the foot of the bed or binding the limbs. Nikethamide 2 c cm intramuscularly every hour is useful for the associated respiratory depression. Solution of adrenaline hydrochloride (1 in 1 000) m 5 every four hours or posterior pituitary extract 0.5 c cm every four hours may be tried but no blood pressure raising drugs are very effective in pneumonia.

Oxygen in high concentrations is very helpful. Unless there is good reason to suspect that the blood volume is diminished filling up the venous system with fluid is not indicated. Little

improvement can indeed be expected until the infection and consequent toxæmia are brought under control. If this is not done circulatory failure will progress to death.

Digitalis is valueless in pneumonia except when auricular fibrillation supervenes. Strychnine should not be used as a routine measure but when there is evidence of failure of the respiratory centre it is worth while to inject slowly a large dose (strychnine hydrochloride gr $\frac{1}{8}$) intravenously dissolved in 5 c cm of saline (See also page 116.)

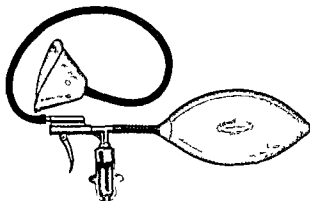


FIG 13

Sparklet apparatus for administration of carbon dioxide

HICCOUGH

Hiccough is a sudden involuntary contraction of the diaphragm which causes air to be drawn in through a partially closed glottis and to make a characteristic noise. When it is rapid and persistent it may call for urgent treatment. This will include treatment of the cause be it abdominal (peritonitis) thoracic (pleurisy) cerebral (encephalitis) or toxic (uræmia). Homely remedies such as breath holding and drinking cold water having failed symptomatic relief may be obtained from one of the following —

- 1 Oil of cloves on a lump of sugar or a stronger carminative such as —

R	Menthol	7½ gr
	Compound spirit of ammonia) 1 fl oz of each
	Spirit of chloroform	
	Tincture of ginger	

One teaspoonful in water taken as strong as possible

- 2 Gastric lavage followed by an alkaline powder
- 3 Hyoscine hydrobromide gr $\frac{1}{100}$ subcutaneously
- 4 Glyceryl trinitrate gr $\frac{1}{100}$ as a tablet under the tongue
- 5 Inhalation of amyl nitrite

It must be admitted that in severe cases these measures are disappointing. Administration of CO₂ in high concentrations via a mask may be effective at any rate for an hour or so. A Sparklet apparatus is convenient (Fig 13). Two other measures have been recommended namely 10 c cm of 10 per cent calcium gluconate intravenously and pulling the base of the tongue forwards with a tongue depressor.

When medical measures fail surgical exposure of the phrenic nerve and injection with procaine is advisable. It will often abolish or greatly mitigate hiccough. A ligature should be passed under the nerve so that it may be easily found again for further injection or avulsion. Because the left diaphragm has the greater excursion it is best to inject the left phrenic nerve.

Even this will sometimes fail and clonic contractions of respiratory muscles other than the diaphragm may go on after bilateral phrenic crushing. Arguing that abnormal afferent impulses were passing up the vagus my colleague Libero Fatti injected 1 minim of alcohol into each vagus nerve in a patient who hiccoughed for six days. Relief was prompt and permanent and the resulting laryngeal paralysis only transient.

C ALLAN BIRCH

CHAPTER VIII

Cardio-Vascular Emergencies

EMERGENCIES are of common occurrence in cardio vascular disease. They range through all grades of severity from the annoyance of nocturnal extrasystoles sufficiently numerous to provoke alarm and prevent sleep to the most profound degrees of pain shock and collapse. Death may threaten in a matter of seconds or minutes. The doctor in consequence may know little or nothing of his patient until faced with the actual emergency. It demands a quick eye and a ready judgment to assess the seriousness of the situation.

A simple classification of the common circulatory emergencies based on the predominant symptom is therefore useful. Admittedly such a system has its imperfections and must be used with discretion as one symptom may dovetail with another. The pain of a coronary thrombosis for instance may be masked by the urgent dyspnoea of left ventricular failure. Conversely an attack of paroxysmal tachycardia—a common cause of palpitation—may be sufficiently severe in the middle aged or elderly to cause angina or even syncope. Palpitation may then be of less significance to the patient. These are exceptions. To be appropriate immediate treatment must be based on underlying causes which unfortunately are sometimes difficult to determine in the urgency of the situation. As a general rule the leading symptom justifies such a classification as the following—

- (1) Syncopal attacks—(a) simple faint (b) carotid sinus syncope (c) postural hypotension (d) Gower's syndrome (e) Stokes Adams seizure
- (2) Collapse and shock
- (3) Palpitation—(a) extrasystolic irregularity (b) paroxysmal auricular tachycardia (c) paroxysmal ventricular tachycardia (d) paroxysmal auricular flutter (e) paroxysmal auricular fibrillation
- (4) Dyspnoea—(a) acute left ventricular failure (cardiac asthma) (b) acute right ventricular failure (pulmonary embolism) (c) cardiac tamponade

- (5) Precordial pain—effort angina and coronary thrombosis
- (6) Peripheral embolism

SYNCOPAL ATTACKS

Syncopal attacks arise as a result of acute cerebral anæmia. The essential feature is an abrupt decrease in the volume of blood in active circulation. A profuse hæmorrhage produces symptoms which differ only quantitatively from those of a simple faint. In the first instance the blood is lost to the exterior; in the second it is temporarily pooled in the venous reservoirs of the splanchnic area.

(a) A simple faint is best treated by loosening the clothing about the neck and by placing the patient flat on his back. This facilitates the return of blood to the heart and tends automatically to correct the cerebral anæmia. An even better effect may be obtained by supporting each foot some inches from the ground thereby allowing gravity to aid the return of blood to the heart. Consciousness is usually regained rapidly. Massage from the periphery towards the heart has a similar effect. Warmth aids recovery. Hot tea or coffee are generally acceptable and are probably more serviceable than whiskey or brandy.

Prevention depends on the detection of exciting causes. Emotional stress in certain excitable individuals should be avoided. Pain precipitates attacks in other people. Long hours without food, the erect posture, a hot stuffy atmosphere and disagreeable sights or sounds are factors known to upset vaso motor control and produce a simple faint.

(b) Carotid sinus syncope is a special variety of fainting attack attributable to a hypersensitive state of the carotid sinus reflexes. That gentle pressure with the finger tips over one or other carotid sinus can reproduce constantly in a matter of seconds a train of symptoms terminating in a syncopal attack, exactly similar to that to which the patient is liable, provides good evidence of the hypersensitive state of the sinus mechanism and serves to distinguish it from other forms of syncope. Pronounced cardiac slowing with or without a fall in blood pressure is a common feature. Adrenaline (0.25 to 0.5 c.c.m.) subcutaneously is a very effective remedy. A small tumour or gland in the neck may be a source of irritation responsible for the attacks and require removal. Section of the sinus nerve or the stripping of the coats of the carotid artery at its bifurcation

on the side of the more sensitive sinus are procedures which have been employed in these patients. Fortunately hypersensitivity of the carotid sinus is not necessarily permanent and measures designed to improve the patient's general health are often effective.

(c) **Postural hypotension** causes syncope very readily when the erect posture is assumed. Postural fainting is still apt to occur even after injecting adrenaline which though it increases the heart rate does not restore vasomotor control in these patients. Ephedrine in 50 mgm doses by mouth even as often as every two or three hours does not always prevent attacks. Anæmia and syphilitic infection of the central nervous system are two common associates of this form of syncope and as such both may require energetic treatment.

(d) **Gower's syndrome** Often confusedly designated a heart attack this condition has for its leading symptom a sense of impending death associated with profound weakness and exhaustion faintness slight breathlessness and tingling of the extremities perhaps of 10 to 30 minutes duration. It causes great alarm to the patient who though distressed does not lose consciousness and does not look seriously ill. The attack is probably a transient disorder of the autonomic nervous system and is commonly produced by prolonged mental strain or anxiety.

In treatment the first step is reassurance. Rest in bed is essential usually for some days after the acute phase has subsided. Sedative drugs such as bromides gr 10 three times a day or phenobarbitone gr $\frac{1}{2}$ twice or three times a day can be employed with advantage. Particular emphasis deserves to be placed on the provision of adequate sleep. A rapidly acting barbiturate such as a tablet of cyclo-barbitone (Burroughs Wellcome & Co) gr 3 followed by bromide gr 20 is a useful and effective combination.

(e) **Stokes Adams seizures** are syncopal attacks associated with a defect transient or permanent in the conducting mechanism of the heart beat. The milder attacks in which the *pause between successive ventricular contractions does not exceed six or eight seconds* cause no more than a momentary sense of insecurity and giddiness. A longer pause is followed by sudden loss of consciousness and in the worst attacks respiratory arrest and convulsions occur.

Recovery from the attack coincides with the resumption of the ventricular action. As a rule this occurs before any particular treatment can be administered. In the most severe attacks injections and inhalations are valueless as the circulation is in abeyance. In desperate cases an intra cardiac injection of 0.25 c cm of adrenaline hydrochloride (1 in 1 000) may be employed (see page 14). Ventricular action is restored immediately as shown by the flush of blood through the skin of the face. This is an emergency measure not without danger and is only justifiable for urgent situations such as a severe Stokes Adams seizure and the cardiac arrest of anaesthesia, electrocution, anaphylactic shock, drowning and asphyxia neonatorum.

Rarely Stokes-Adams seizures recur at short intervals, the patient regaining consciousness only to relapse into coma every few minutes. In such circumstances adrenaline 0.5 c cm by subcutaneous injection is a valuable remedy. The beneficial effect becomes apparent in 10 or 15 minutes by which time the seizures may stop. If they persist the dose should be repeated.

When attacks occur sporadically at longer intervals ephedrine gr $\frac{1}{2}$ to 1 by mouth every four to eight hours may ward off attacks by increasing the excitability of the ventricular muscle. The dose should be no more than that necessary to increase the ventricular rate under resting conditions by three or four beats per minute. When ephedrine fails a trial may be made with one of the slow acting adrenaline compounds such as adrenutol (Evans) 1 to 2 c cm or adrenaline in oil (Parke Davis & Co) (page 95) 1 to 2 c cm by subcutaneous or intramuscular injection once or twice daily. Barium chloride $\frac{1}{2}$ to 1 grain in a mixture thrice daily by mouth has an experimental justification for its use in that it is said to heighten the excitability of the cardiac muscle but clinical experience fails to substantiate this claim. Atropine ($\frac{1}{50}$ to $\frac{1}{33}$ gr) subcutaneously is a remedy commonly employed. It is of little or no value.

Rarely recurrent Stokes Adams seizures are the result of the transient interruption of co-ordinated ventricular contraction by short phases of ventricular fibrillation. This state of affairs can only be recognised with certainty by the use of the electrocardiograph. It should be suspected when multiple seizures fail to respond to ephedrine or adrenaline. When ventricular fibrillation is responsible for the Stokes Adams attack it is justifiable

to prescribe quimidine sulphate (0.2 gm. in capsules or cachets) at four-hourly intervals. The dose can be increased or reduced in amount according to circumstances.

COLLAPSE AND SHOCK

There is no fundamental difference between "medical" collapse and "surgical" shock. The underlying mechanism is a decrease in the amount of blood in *active* circulation as a result of a disorganisation of the peripheral circulation. The ashen grey colour, the restlessness, the exhaustion, the cold damp skin, the almost imperceptible pulse, the shallow respirations and the impaired mental acuity, make a characteristic picture often dramatic in its rapid development. In medical experience collapse is associated chiefly with acute infections such as the most virulent forms of lobar pneumonia (*page* 109), with profuse hæmorrhage as from a bleeding peptic ulcer (*page* 60) or perhaps most commonly in connection with tissue infarction, notably the myocardial infarct of coronary artery occlusion. The essential features common to all varieties are a reduction in the volume of circulating blood, a fall in blood pressure, impaired oxygenation of the tissues, and a progressive bodily enfeeblement.

Treatment—The classical remedies for shock are posture, warmth, morphine and reassurance.

(a) *POSTURE*—While the patient's condition is being assessed and preparations made for the major methods of treatment, attention should be devoted to nursing care. Posture is important. Unless otherwise contra-indicated, the patient should be encouraged to lie flat in bed, pillows and supports being withdrawn from the head. The foot of the bed should be raised on blocks, one to two feet high, to facilitate the return of blood to the heart and brain.

(b) *WARMTH*—Since cold increases shock, a generous supply of hot bottles, or the application of a 'shock cage' in which heat is maintained by electric bulbs, is of value. The hands and feet must be kept warm. Similarly the provision of hot drinks is valuable in correcting dehydration and giving warmth. Hot soups, hot normal saline with glucose, strong tea or coffee may all be administered with benefit.

(c) *MORPHINE*—Pain is a potent factor in perpetuating those

reflexes which disorganise the peripheral circulation and promote the symptoms and signs of collapse. Morphine is well tolerated in the early stages and may be given in full doses hypodermically. After some hours when the disorder of the peripheral circulation is more advanced the degree of shock more profound and the patient more enfeebled smaller doses of morphine ($\frac{1}{8}$ to $\frac{1}{6}$ gr.) are justifiable if only to reduce restlessness and give peace of mind. The detrimental effect of pain is to be dreaded more than the peripheral reaction to this drug and there can be few instances in which morphine is not indicated for a shocked patient.

(d) REASSURANCE.—It is also the duty of the physician to do what he can to counter the sense of apprehension and the fear of impending disaster—commonly associated with even minor grades of shock. Reassurance and encouragement mean much to shocked patients.

Other methods of treatment are valuable in special cases—

(e) TRANSFUSION.—War experience has amply confirmed the view that the most effective therapeutic agent in counteracting the essential deficiency in shock or collapse is the restoration of an effective blood volume. When shock results from massive hæmorrhage transfusion of whole blood is manifestly of prime importance. If suitable typed blood is not immediately available various substitutes may be used but whole blood is preferable. It may be given from a suitable container into a vein at a rate of 100 to 200 c cm per hour for 12 to 24 hours or even longer depending on the patient's progress and general reaction. In assessing the situation blood pressure readings are of the greatest importance. A fall in systolic pressure below 100 mm is a danger signal and usually indicates the necessity for continued transfusion.

In the absence of matched blood or when replacement by blood itself is not essential the restoration of a satisfactory circulatory volume can be brought about by normal saline 5 per cent glucose in normal saline or reconstituted mixed plasma. These agents lack the oxygen carrying capacity of blood and while saline and glucose are helpful in an emergency they do not provide the colloid osmotic pressure of plasma which maintains fluid within the circulation. Synthetic plasma substitutes are under trial. Gum acacia in 6 per cent solution has approximately the same viscosity as plasma. It exerts an osmotic

pressure and may be used in amounts up to 500 c cm in saline intravenously

(f) OXYGEN (*See page 409*)—Of the various remedies used in the treatment of circulatory collapse oxygen is secondary in importance only to morphine. Anoxæmia has a detrimental effect on both the central and vegetative nervous systems and thus aggravates all the features of shock. It is of particular value in those forms of anoxæmia in which œdema of the lung alveoli interferes with the absorption of oxygen. Pulmonary and myocardial infarction are the two conditions in which oxygen is of particular value whether cyanosis be present or not.

(g) ANALGESICS—Synthetic compounds such as nikethamide (pyridine carbonate of diethylamide) or leptazol (pentamethylene tetrazol) appear to be useful and effective remedies for cardio-respiratory failure in doses of 1 c cm or more intramuscularly at one or two hourly intervals. Much larger doses (10 c cm in 250 c cm of saline by intravenous drip) may be used in circulatory collapse when the critical situation justifies desperate remedies. It is doubtful if either of these drugs is active by mouth. They may be used alternately with strychnine by two-hourly intramuscular injections over the acute phase of respiratory failure in pneumonia, septic peritonitis or acute delirium tremens. They are also useful for the mild degree of collapse inseparable from full and repeated morphine administration.

Strychnine has a reputation in the treatment of the central respiratory depression of lobar pneumonia. It may be given in doses of $\frac{1}{3}$ gr subcutaneously at two or four hourly intervals. But strychnine is a cumulative drug and three or four such doses usually produce a well marked tendency to tetanic spasms easily demonstrable by eliciting the tendon reflexes. Administration should not be continued beyond this stage. The value of strychnine for circulatory failure unassociated with respiratory failure is very doubtful.

(h) EXTRACT OF SUPRARENAL CORTEX—While the ideal drug for the treatment of acute peripheral circulatory failure has not yet been found, extract of supra renal cortex B.P.C. by correcting the abnormal capillary and cellular permeability is believed to control the volume of circulating fluid. It may be added to the transfusion fluid in the proportion of 2 c cm to each litre. The demonstration that a renin-like substance set

taken digitalis or a related substance within the preceding 10 days. Further, it is desirable to be certain of the nature of the paroxysm before digoxin is used. This necessitates electrocardiographic observations. If it is known that the paroxysm is *not* of the ventricular variety, digoxin can be employed with justification. Given intravenously in a dose of 1.0 to 1.5 mgm suitably diluted in 10 or 20 c cm of normal saline digoxin by its vagal stimulant properties is capable of arresting auricular tachycardia often within 10 or 20 minutes of the completion of the injection.

(vii) **QUINIDINE**—If digoxin fails and the patient's condition is deteriorating, quinine or quinidine may be tried. The oral route is preferable, using quinidine sulphate in capsules or cachets in doses of 0.2 to 0.4 gm at four hourly intervals. Alternatively quinidine sulphate, 0.3 gm in 10 to 20 c cm saline may be *slowly* injected intravenously, preferably under electrocardiographic control.

(c) **Paroxysmal ventricular tachycardia** is usually associated with organic heart disease. It may be an alarming and serious complication of coronary artery thrombosis, or may arise as a manifestation of digitalis intoxication in elderly patients.

Treatment—The first step is to give a sedative—bromide barbiturate, or morphine, depending on the urgency and duration of the symptoms. Ipecacuanha, mechoLIN and digoxin and mechanical methods of vagal stimulation are valueless and contra indicated. Quinidine is the drug of choice. A suitable scheme is to order 0.2 gm by mouth at four hourly intervals with the omission of the 2 a.m. dose so as not to disturb sleep. The dose can be continued safely for 36 to 48 hours if the attack persists. Larger doses can be used preferably under electrocardiographic control. Usually the ventricular rate slows progressively under the influence of quinidine until it is around 90 to 110, when the abnormal rhythm ceases abruptly.

When conditions are more urgent the patient's distress increasing, and portal and peripheral congestion complicating the situation, it may be considered expedient to use quinidine intravenously, preferably by continuous intravenous drip. Too rapid injection may cause serious circulatory collapse. By vigorous shaking, 50 to 60 grains of quinidine sulphate can be dissolved in 500 c cm of a 5 per cent glucose solution. After filtering and

warming the sterile solution is run in at a rate of 1 to 2 c cm per minute until normal rhythm returns or until cinchonism shown by headache roaring in the ears and some degree of deafness is induced. In my experience this is the safest and most effective method of administering quinidine intravenously. It seldom fails to restore normal rhythm.

(d) **Paroxysmal auricular flutter** resembles paroxysmal auricular tachycardia and may be distinguished by the temporary slowing and irregular action induced by carotid sinus stimulation. It is probably the rarest of the acute paroxysmal disorders and gives rise to less trouble in that the heart rate is seldom over 160 beats a minute and individual attacks are of short duration.

Treatment—Digitalis is indicated. It is seldom necessary to use the intravenous route but if the rhythm causes distress and attacks occur with sufficient frequency it is wise to allow the patient to have a supply of a reliable digitalis preparation such as digoxin which can be taken in a full dose by mouth at the onset of the attack. Providing that none has been taken the preceding week 10 to 15 mgm (i.e. up to six 0.25 mgm tablets) can be taken at once. Dried digitalis leaf is equally if not more efficient but as it is excreted more slowly than digoxin full dosage must not be given until two weeks have elapsed since the last dose of digitalis so as to avoid intoxication. Rapid digitalis effects may be produced by a full dose of 15 to 20 grains of the powdered leaf in divided amounts over a 24 hour period. Digitalis breaks up the regular ventricular rhythm of auricular flutter converts flutter to fibrillation and may bring about a reversion to normal rhythm. On account of the danger of doubling the ventricular rate by the abolition of block quinidine is better not used in the treatment of paroxysmal flutter.

(e) **Paroxysmal auricular fibrillation**—This is much more commonly encountered than auricular flutter. After operation in elderly people in the course of acute infections such as pneumonia and particularly in thyrotoxicosis bouts of auricular fibrillation are common. Distress results from the excessive rate the thumping irregular beats and occasionally from a choking sensation in the throat.

Treatment—When subjective distress is obvious or when signs of heart failure appear imminent treatment with digitalis is desirable. The object is to bring the grossly excessive rate

under control. This can be done either by using digoxin or digitalis leaf as described for auricular flutter. The heart rhythm usually reverts to normal within a few days of digitalisation. Quinidine is reserved for those patients in whom digitalis fails.

DYSPNŒA

(a) **Acute left ventricular failure.**—Extreme dyspnœa occurring at rest, often during the night, perhaps after a strenuous day, is a symptom of considerable consequence (*See also page 85*). Occurring in a paroxysmal form, and preceded by a persistent tickling cough, with increasing restlessness, it is a sign of a failing left ventricle, commonly the result of long continued hypertension, aortic stenosis or insufficiency, a myocardial infarct, or less commonly a tight mitral stenosis. Unless relieved, the urgent dyspnœa advances to acute pulmonary œdema with the production of white, and later pink, frothy sputum. Such a condition arises primarily as a result of a disproportion in the ventricular outputs, the left being unable to deal with all the blood fed into the pulmonary circuit by the right side of the heart.

Treatment—The objects of treatment are to allay restlessness, increase the efficiency of the left ventricle, and to reduce temporarily the output of the right ventricle.

The patient prefers to sit bolt upright, supported by pillows, with the feet dependent. He must be kept warm, reassured, and given morphine $\frac{1}{4}$ to $\frac{1}{2}$ gr subcutaneously at once. By this means his strength is conserved, the exhausting cough relieved, the venous return reduced, and dyspnœa eased.

A venesection is the next step (*page 421*) and in the presence of white or pink sputum there should be no hesitation in performing it. In less urgent cases the venous return can be reduced by the application of sphygmomanometer cuffs, bandages, tourniquets or towels to the proximal portion of the thighs and arms at a pressure only a little in excess of the diastolic blood pressure. In this way blood may be temporarily shunted from the right side of the heart. Compression of the limbs is continued from 10 to 20 minutes and then gradually released from one extremity after another at intervals of a few minutes. The whole procedure can be repeated if necessary. With similar intent, the administration of nitroglycerine has been

recommended. A tablet of glyceryltrinitrate $\frac{1}{100}$ gr allowed to dissolve under the tongue causes dilatation of the splanchnic vessels and temporarily reduces the venous return to the heart and theoretically should lessen the right ventricular output. As soon as practicable oxygen should be administered.

When attacks are severe and the response to the above measures less satisfactory than might be anticipated a full dose of digitalis should be administered provided that no allied preparation has been taken in therapeutic amounts in the preceding two weeks. For a prompt effect there is no better preparation than *digovin* in a dose of 1.0 to 1.5 mgm suitably diluted in 10 or 20 c cm of saline by the intravenous route. It should be noted that digitalis and nitroglycerin are better avoided when the clinical findings suggest that the urgent dyspnoea arises as a result of an acute coronary occlusion. In these circumstances theophylline and ethylene diamine (aminophyllin) in a dose of 0.25 to 0.5 gm in 50 c cm of 10 to 20 per cent glucose by slow intravenous injection may be used instead of digitalis during the acute phase of the illness. Similarly there should be no hesitation in using a mercurial diuretic. Mersalyl B.P. 1 to 2 c cm may be given intramuscularly or intravenously or Neptal 1 to 2 c cm intramuscularly. A specially prepared solution of Neptal may be used intravenously in doses of 5 c cm. These are potent agents for the relief of the pulmonary oedema of left ventricular failure.

(b) **Acute right ventricular failure** results from acute dilatation of the pulmonary artery and right ventricle caused by the impaction there of a massive embolus. (See also page 99.) When this is very large and obstructs one or other pulmonary artery death results almost instantaneously. In less severe instances there is intense dyspnoea, thoracic oppression, substernal pain and the rapid development of circulatory collapse, the whole picture bearing a striking resemblance to coronary thrombosis from which diagnosis may be difficult (see page 100).

Morphine should be used liberally $\frac{1}{4}$ to $\frac{1}{2}$ gr subcutaneously and oxygen administration started at once. By this means dyspnoea, cyanosis, pain and anxiety can be eased appreciably and shock lessened. Venesection is to be condemned as it depletes further a circulation already hampered by the disorganisation of peripheral circulatory failure. Indeed a falling blood

pressure might be taken as an indication for a glucose saline transfusion. Nikethamide or leptazol by subcutaneous injection at two-hourly intervals in a dose of 10 to 15 c cm is often helpful in warding off the harmful effects of a profound degree of collapse which can develop rapidly. Atropine $\frac{1}{100}$ gr combined with papaverine hydrochloride $\frac{1}{2}$ to $\frac{3}{4}$ gr by the intravenous route is said to ease the situation by the relaxation of spasm in the pulmonary and coronary vessels. Finally the heroic procedure of embolectomy has been attempted with success in rare instances (page 101).

(c) **Cardiac tamponade**—This term is applied to the combination of symptoms and signs which result from an increase in intra pericardial pressure sufficient to interfere with the venous return to the heart. Excessive amounts of fluid within the pericardium may accumulate rapidly and unless the pericardial sac stretches sufficiently the great veins are compressed the venous inflow retarded and the ventricular outputs reduced. A mechanical reduction of the intra pericardial pressure may be considered desirable during the course of rheumatic pericarditis if it be judged that the effusion is accumulating with such rapidity as to lead to venous engorgement in the neck and a progressive fall in blood pressure. It is said that the best indication for withdrawal of pericardial fluid is a pulse pressure below 20 mm. Day to day blood pressure records in pericarditis with effusion are of considerable value. Tapping the pericardium is a procedure only to be undertaken as a last resort when orthopnoea is severe thoracic and epigastric oppression a real embarrassment and when the pulse pressure is falling steadily. Most cases of pericardial effusion of rheumatic origin make a good recovery without tapping. It is more often necessary in tuberculous or malignant pericarditis septic infection of the sac or on account of bleeding from wounds of the heart.

In rheumatic pericarditis with a large effusion a safe measure is to puncture the pericardium from behind (*see page 381*).

PRÆCORDIAL PAIN

Effort angina and coronary thrombosis—The fully developed picture of a major coronary thrombosis is well known—prolonged pain and collapse followed by a minor fever and leucocytosis being the outstanding features. There are all grades of severity.

so that on occasion it may be difficult to distinguish the effort pain of a transient attack of angina pectoris from a minor coronary thrombosis. If the first attack of angina of effort is sufficiently severe to warrant an emergency call it is justifiable for all practical purposes to regard it as an attack of coronary thrombosis. Treatment should be planned accordingly. Similarly should anginal attacks in a known subject of coronary disease suddenly increase in frequency and severity and particularly if pain should be experienced at rest—angina decubitus—the doctor should suspect that a local thrombosis is imminent and treat as for coronary thrombosis.

Treatment—The objects of treatment are to counteract shock to limit the size of the resulting infarct and to promote a firm fibrosis in it.

The more the local oxygen requirements exceed the coronary supply the larger is the area infarcted. Complete rest reduces myocardial activity to a minimum limits the area infarcted conserves energy and favours healing. Morphine $\frac{1}{4}$ to $\frac{1}{2}$ gr repeated in sufficient quantity to abolish pain also lessens shock and allays anxiety. The patient should therefore be put to bed at once and the head raised on one pillow unless dyspnoea is troublesome when the shoulders may be supported.

Warmth should be maintained by hot bottles and hot drinks. Visitors should be excluded. At this stage of the illness if circumstances permit the patient is better kept at home than moved to a hospital at least until the initial shock has subsided and the pain is adequately controlled. Full advantage must be taken of rest in bed. For some days until the acute phase of the illness is passed the patient must do nothing for himself. He must be fed, moved and nursed as gently as possible. He should not be given purgatives even for three or four days.

Oxygen therapy is of great value even in the absence of cyanosis particularly in the early stages. It helps to relieve pain limits the myocardial damage and reduces the degree of shock.

It is seldom that other remedies are necessary in the acute phase. If an attack of so called angina of effort is so severe and protracted as to suggest the use of amyl nitrite it is probable that a coronary occlusion has occurred in which case morphine is the drug of choice. For emergency use amyl nitrite is better

abandoned. By lowering the blood pressure it accentuates the degree of shock, is believed to increase the area infarcted, and seldom controls the pain. Nitrites have their most useful function in the *prevention* of effort pain when, as tablets of glyceryl trinitrate $\frac{1}{200}$ to $\frac{1}{50}$ gr., they are allowed to dissolve slowly under the tongue in anticipation of pain. Nitrites should not be used in circumstances in the least suggestive of myocardial infarction. Aminophylline, 0.25 gm. intravenously, may be used as an adjunct to morphine for the relief of pain, and nikethamide or leptazol at two or three hourly intervals may help to counteract the tendency to circulatory collapse.

Rest in bed for six weeks and a gradual return to activity serve to promote a firm myocardial scar and reduce the likelihood of subsequent effort pain.

PERIPHERAL EMBOLISM

Most arterial emboli are derived from the heart, thrombi becoming detached from the valves, as in bacterial endocarditis or from the endocardial lining as in mitral stenosis or myocardial infarction. The lodgment of a clot in a peripheral artery obstructs the local blood supply, often produces an intense vascular spasm throughout the limb distal to the clot, and gives rise to local anoxæmia manifesting itself by numbness and coldness of the extremity, a more or less cadaveric appearance of the limb, and often by considerable pain throughout the affected area. The oft quoted alliterative description of symptoms "pain, pallor and paralysis" is somewhat misleading since pain occurs in perhaps only 50 per cent. of cases. If the local circulatory deficiency be not made good by adequate collateral channels, or by the timely removal of the clot from the major vessel, the tissues perish and at a level varying with the site of the arterial block, a line of demarcation forms distal to which the limb becomes gangrenous.

In the arm on account of the rich blood supply, embolectomy is seldom required, but when a lower limb is affected it is wise to assume at the outset that surgical removal of the clot will be necessary within an hour or two. For operative success the general condition of the patient must warrant interference and the procedure must be undertaken without unnecessary delay. Experience has shown that the longer the embolus

remains impacted the greater the local injury to the arterial wall, and that even after its removal, thrombosis on the damaged intima is prone to occur and may spread peripherally. To be successful, embolectomy should be undertaken within 10 hours of the impaction of the clot. Prompt measures can often save the limb. Allen, from a large experience, emphasises three important "don'ts." Don't delay, don't elevate the limb, and don't overheat it.

DON'T DELAY—Conservative measures are important not only in those patients in whom an operation is contra-indicated, but in those for whom surgery cannot be immediately undertaken. Indeed, the medical measures now advocated may prove in themselves so satisfactory that surgical help may not be necessary, but a decision must not be postponed too long. If appropriate medical measures have not produced a *striking* improvement in the state of the limb *within three or four hours of the lodgment of the clot* then embolectomy should be attempted immediately.

DON'T ELEVATE THE LIMB—The object in treatment is to increase the blood supply. This can be done mechanically by elevating the head of the bed on blocks when the leg is involved, or by having the patient sit up in bed with the arm dependent if the brachial artery is the site of the impaction.

DON'T OVERHEAT THE LIMB—Heat is essential in treatment but should not be applied directly to the affected extremity. It is a common mistake to surround the impoverished limb with hot water bottles whose temperature often exceeds 150° F (65.6° C). By increasing the local metabolism, excessive heat accentuates the nutritional deficiencies, aggravates the tendency to gangrene and commonly provokes extensive burns of the devitalised skin. The entire limb should be wrapped in warm cotton wool held lightly in place by a bandage, thus preserving its natural temperature and protecting it from the ill effects of excessive heat. A cradle containing one or not more than two, light bulbs preferably left open at one end may be placed over the limb but the temperature of the air within the cage should not exceed 105° F (40.5° C).

Learmonth has shown how effective reflex vasodilatation may be in increasing the blood supply to the affected limb. By heating the hands, blood flow in the foot and leg may be enhanced. A warm bed jacket should be worn to protect

the arms from chilling and with one hand in a box or under a cradle, heated by a light bulb the temperature may be raised sufficiently high to induce local sweating. A glove should be worn on the opposite hand. By this means vasoconstriction in the foot and leg may be released reflexly.

If pain is severe opiates should be given generously. Morphine $\frac{1}{2}$ to $\frac{1}{4}$ gr may be administered hypodermically or if pain is less intense adequate control can be obtained by the repeated use of a compound tablet of aspirin phenacetin and codeine taken by mouth as necessary. Papaverine hydrochloride has valuable anti spasmodic properties and is of great value on this account when given intravenously in a dose of $\frac{1}{2}$ to $\frac{3}{4}$ gr. Alcohol in the form of whiskey or brandy has valuable vasodilator properties when taken by mouth and may therefore be prescribed generously. Spinal anæsthesia is a further procedure which can be employed to promote maximal but temporary vaso-dilatation when the lower limb is involved.

If it be judged that the patient is unfit for embolectomy or if more than 10 or 12 hours have elapsed since the embolism the area of the limb liable to the development of gangrene must be kept clean and dry by the application of surgical spirit and sterile dusting powder once or twice daily. Amputation can be undertaken at a later date if the patient survives.

A. RAE GILCHRIST

CHAPTER IX

Emergencies in Blood Diseases

WHEN it is suggested that a blood disease may be responsible for urgent symptoms it is of the greatest importance that at least a provisional diagnosis should be made before commencing treatment no matter how acutely ill the patient may be. Inappropriate treatment may waste time and particularly in megalocytic anæmias render the diagnosis more difficult. Although the history may be suggestive in some cases the symptoms are often too varied to be more than pointers to possible diagnoses.

A full blood count should therefore be made and should include the following procedures —

- (a) hæmoglobin estimation
- (b) erythrocyte count
- (c) leucocyte count
- (d) examination of a blood film and differential white cell count
- (e) reticulocyte and platelet counts

Bone marrow examination should be included if indicated.

In severe anæmia and in all cases where the condition has been caused by hæmorrhage it is advisable to determine the patient's blood group as soon as possible and put in hand all necessary arrangements for carrying out transfusion at short notice.

The blood diseases we shall consider from the emergency point of view are —

The anæmias

Polycythæmia

Leukæmias

Agranulocytosis

Purpura hæmophilia and other hæmorrhagic diseases

ANÆMIAS

Diseases causing diminution of the red cells and hæmoglobin can be grouped according to their causation as follows

- (1) Loss of blood which may be acute or chronic continuous or intermittent e.g. following injuries and internal hæmorrhages

- (2) Aplasia or hypoplasia of the bone marrow (normocytic normochromic anæmia), *e.g.* in aplastic anæmia or associated with acute leukæmia, thrombocytopenic purpura or severe sepsis
- (3) Deficiencies in red cell formation from faults in the gastro hæmopoietic mechanism (macrocytic hyperchromic anæmia)
- (4) Deficiencies in red cell formation primarily from faulty iron absorption and metabolism (microcytic hypochromic anæmia)
- (5) Blood destruction as in the various types of hæmolytic anæmia

Post hæmorrhagic anæmias may arise from trauma, long continued slight blood loss from the alimentary or genito urinary tracts, or they may be the consequences of bleeding such as occurs in hæmophilia, severe purpura scurvy and multiple hereditary telangiectasia. The diagnosis is suggested by the history and the general symptoms of anæmia.

The main hæmatological findings are a reduction in hæmoglobin and red cells. Hence at first after an acute blood loss there is a total reduction of all blood constituents with a normal hæmoglobin percentage, red cell count and colour index owing to loss of whole blood. Soon however, with the subsequent hæmo dilution by fluid withdrawn from the tissues, an anæmia develops with a low red cell count and much reduced hæmoglobin percentage. With chronic blood loss, red cell formation may outstrip the available hæmoglobin leading to a hypochromic microcytic anæmia and the colour index may be very low (between 0.3 and 0.6). Sometimes there is also leucopenia. Bone marrow examination is not diagnostic unless the anæmia is secondary to malignant metastases multiple myeloma and such conditions as Gaucher's disease.

Treatment—Bleeding should be stopped, if practicable, by appropriate measures (*see hæmatemesis page 60, hæmoptysis page 93, and epistaxis, page 92*), and adequate rest and warmth must be secured.

If the anæmia is severe (red cells less than 2,000,000 per c mm hæmoglobin less than 40 per cent) and in order to combat shock from severe blood loss, transfusions (at least two to three pints of whole blood) should be given as soon as possible to restore blood volume and raise the falling hæmoglobin concentration.

and red cell count Plasma may be needed if there is much loss of blood volume with low blood pressure and dehydration, and may be given at once while blood is being obtained Ferrous sulphate tablets B P gr 9, three times a day should be prescribed A full diet, especially of protein foods should be taken as soon as possible

Aplastic anæmia.

In this condition the patient is often seen for the first time acutely ill and with a fairly short history of increasing weakness tiredness dyspnœa, palpitation, disinclination to do anything and marked waxy pallor of the skin There has often been an exacerbation in the previous few days or weeks with bruising of the skin and bleeding from the gums nose and mucous membranes

The disease comes on very insidiously between the ages of 15 and 40 as a progressively severe normocytic normochromic anæmia without evidence of regeneration or abnormal cells in the peripheral blood, and a negligible reticulocyte count Sternal biopsy shows a more or less complete aplasia of the bone marrow The spleen is rarely enlarged The secondary types of aplastic anæmias come on at any age after exposure to toxic materials such as benzene, radio active substances, gold arsenic sulphon amides thiouracil amidopyrine etc or as a sequel to severe overwhelming infections or sepsis

Treatment —The patient must be removed from any possibly toxic influences Repeated transfusions of fresh whole blood or preferably concentrated red cells will be required in large amounts It may be necessary to continue these at frequent intervals, often for many months or years Females must have the Rh group determined and be transfused accordingly In the case of males we need not bother about the Rh group in an emergency unless there have been many previous transfusions Much help can be given by the Regional Transfusion Officer

Every endeavour should be made to raise the blood count to normal as soon as possible since this gives longer periods of remission Transfusion usually stops any bleeding from mucous surfaces Many other methods of treatment have been tried, such as giving large doses of liver, iron, extracts of spleen yellow and red bone marrow, folic acid, etc but they are of no value

Ascorbic acid 200 mgm daily and ferrous sulphate tablets gr 9 three times daily should be ordered. Measures should be taken to minimise the special danger of infection to which these patients are liable.

When the emergency is over the patient should be instructed to report regularly for blood counts so that the need for transfusions can be kept under review.

Macrocytic anæmias are characterised by lower red cell counts relative to the hæmoglobin percentage and a preponderance of large cells with consequently a raised colour index. Sternal marrow biopsy discloses a typical megaloblastic hyperplasia. These blood and marrow pictures are seen in all cases where there is impairment of the gastro hæmopoietic mechanism leading to an inadequate supply of the anti-pernicious anæmia liver factor to the bone marrow. The clinical findings vary somewhat according to the place in the hæmopoietic mechanism at which the fault has occurred. This may lie in defective diet, gastric deficiencies (pernicious anæmia or after gastrectomy), intestinal disorders (multiple fistulæ, parasites and sprue), achrestic anæmia or liver disease such as cirrhosis. Consequently treatment will vary somewhat in these different conditions. Of all of them however **pernicious anæmia** is the commonest in temperate climates and is always accompanied by achylia gastrica while the others are usually associated with normal or diminished gastric secretory functions. When fully developed it is usually seen in a patient over 40 who complains of dyspnoea, palpitation, glossitis and ulceration of the mouth and tongue, flatulent dyspepsia, recurrent diarrhoea, paræsthesiæ in the hands and feet or more marked symptoms and signs of postero-lateral cord involvement. On examination there is a lemon yellow skin, prematurely grey hair which is soft and silky in texture and a variable degree of splenomegaly. Retinal hæmorrhage and optic atrophy are sometimes found. Rarely acute abdominal symptoms appear (*see page 46*). Blood examination reveals a severe megalocytic hyperchromic anæmia (with high colour index), polychromasia, anisocytosis and poikilocytosis and a reticulocytosis for about two weeks after active treatment or spontaneous remissions. The bone marrow shows megaloblastic hyperplasia.

Similar symptoms may be noted in achrestic anæmia which comes on between the ages of 20 and 50 and closely resembles pernicious anæmia clinically. The gastric secretion is usually normal and neurological changes are absent. Treatment produces little or no response.

Treatment of uncomplicated pernicious anæmia is relatively easy. A potent liver extract should be used—guaranteed by the makers to have been adequately tested on the human subject. Among British products the following may be mentioned—Hepastab or Hepastab Forte (Boots) Anahepol or Hepolon (Allen & Hanbury) and Pernæmon (Organon Laboratories). 1 to 4 c cm should be given by intramuscular injection daily for two to three days and then 1 to 4 c cm weekly until the blood count is normal.

It should very rarely if ever be necessary to give blood transfusions to patients suffering from pernicious anæmia even in an emergency. When the red count is very low indeed (say less than 1 000 000) some would use 'packed' red cells but such transfusions in these circumstances are not without risk. Repeated transfusions are however necessary in macrocytic anæmias of the achrestic type and in those associated with hepatic cirrhosis since they are resistant to other treatment. They would be indicated if a case of apparent pernicious anæmia did not show a quick response to liver injections by a reticulocytosis.

The place of stomach preparations (*e.g.* Pepsac Boots or Extomac Benger's) in the emergency treatment of pernicious anæmia must be mentioned. They are as effective as liver extracts and should certainly be used if spinal cord and peripheral nerve changes are present and are best given in doses of 10 gm three times daily after meals in *cold* water or milk.

Ancillary treatment for pernicious anæmia by a hydrochloric acid and pepsin mixture, ascorbic acid and an adequate protein intake should be prescribed.

While folic acid 20 mgm daily is effective temporarily in many cases of pernicious anæmia it is not so good as the active liver extracts. It has no value at all in the prevention or relief of neurological complications. There are no indications for its use in the emergency treatment of pernicious anæmia but it may be of value in other macrocytic anæmias.

Hypochromic microcytic anæmia as well as chlorosis and the

Plummer Vinson syndrome (dysphagia with anæmia) occur almost exclusively in women between 20 and 50. A similar anæmia, however, may be seen in either sex as a secondary manifestation of malignant disease, tuberculosis, nephritis, or after gastro enterostomy and gastrectomy.

This type of anæmia develops insidiously and so the patient may not be seen until late in the course of the disease when some symptom such as excessive tiredness, exhaustion, pallor, dysphagia, loss of appetite and wasting makes her seek advice urgently.

Other non urgent symptoms of help in diagnosis are flatulent dyspepsia, intermittent diarrhoea, angular stomatitis, soreness of the mouth and tongue, dry, coarse, and brittle hair, spoon shaped finger nails (koilonychia) and menstrual disturbances.

Examination reveals pallor of the skin and mucous membranes. There is rarely any splenomegaly. Blood examination discloses a hypochromic microcytic anæmia with often a very low colour index (*e.g.* 0.3 to 0.4). There is no evidence of hæmolysis. The bone marrow is normoblastic in type. Achylia or achlorhydria is commonly found.

Treatment—Blood transfusion is rarely needed and only if the hæmoglobin is very low—say below 25 per cent. If it is used, care should be taken to avoid circulatory overloading (*see page 30*).

Ferrous sulphate tablets B.P. gr. 9, three times a day should be given—smaller initial doses being used for patients who do not tolerate iron well. Iron in mixture form (iron and ammonium citrate gr. 40, three times a day) is necessary when there is dysphagia. Intravenous injections of complexes of the sucrose iron type (Ferrivenin, Bengel) are of value in those patients intolerant of or failing to respond to iron by mouth.

Other treatment which should be ordered includes ascorbic acid 200 mgm. daily, and a hydrochloric acid and pepsin mixture. If glossitis or koilonychia are marked it is wise to give a vitamin B preparation (Becosym, Roche, three to six tablets daily).

Hæmolytic anæmias all show the features common to any anæmia, together with a varying degree of jaundice. We are concerned here only with the acute types such as the hæmolytic anæmia of Lederer, hæmolytic crises in acholuric jaundice, erythroblastosis foetalis (*see page 261*), and hæmolytic anæmias

from known causes (*e.g.*, gold, arsenic, benzene, etc.), malaria (*see page 282*), and Marchiafava's paroxysmal hæmoglobinæmia with hæmoglobinuria. Occasionally acholuric jaundice is complicated by biliary colic caused by pigment stones (*see page 46*).

Treatment—Rest in bed and removal of toxic hazards are clearly indicated. If arsenic (and possibly other metals) are responsible, the use of British Antilewisite (BAL) should be considered (*see page 238*). Sodium thiosulphate 0.4 to 0.6 gm. as the B.P. 10 per cent. solution should be given intravenously for gold intoxication.

Repeated blood transfusions are usually needed. The remarks about the Rh factor under aplastic anæmia (*page 134*) apply here also. Splenectomy is the best treatment for acholuric jaundice, but is rarely needed as an emergency measure. In Marchiafava's hæmolytic anæmia intensive alkalimisation of the urine is important. Potassium citrate or sodium bicarbonate in doses of 60 gr. should be given three hourly.

POLYCYTHÆMIA

This disease usually presents in a patient over 45 with severe headaches and sometimes with painful flushing of the skin of the legs. The skin, especially of the face, is dark red or plum coloured. There is a variable degree of splenomegaly. The diagnosis is therefore obvious before complications arise. Occasionally the presenting feature is cardiac failure, hyperpæsis or even cerebral hæmorrhage or thrombosis. Treatment is then purely symptomatic and venesection is the method of choice for cardiac and hypertensive symptoms. Remedies for the primary blood condition such as X-ray irradiation, phenylhydrazine or radio active phosphorus are not urgently called for but must be considered in the prevention of recurrence of urgent symptoms.

While achlorhydria is usual in polycythæmia, a high gastric acidity is occasionally found. This may be associated with symptoms of duodenal ulceration which may complicate the picture and call for additional measures.

LEUKÆMIAS

Chronic myeloid and lymphatic leukæmias may present as emergencies because of the weakness and dyspnœa they cause

Acute abdominal pain may result from perisplenitis. Marked splenomegaly is practically a constant finding in chronic myeloid leukaemia and some degree of enlargement of the liver is common. Glandular enlargement is normally seen to a greater degree in chronic lymphatic leukaemia.

These clinical findings suggest the diagnosis which is confirmed by the high total white count (up to 600,000 or 800,000 per cu mm) with corresponding changes in the differential count according to the type. In myeloid leukaemias, myelocytes, promyelocytes and myeloblasts are found and, in lymphatic leukaemia, lymphocytes and lymphoblasts.

In acute leukaemia and monocytic leukaemia the problem is less easy. They are more commonly seen in children and young adults. The onset is often insidious and the symptoms protean. There is little or no splenic enlargement and, in the early stages, the peripheral blood picture is often more suggestive of pernicious anaemia, agranulocytosis or aplastic anaemia than a leukaemia. A sternal marrow biopsy will usually give the correct diagnosis even in the early stages.

Treatment—Except in the terminal stages of chronic leukaemia, transfusion of whole blood will improve the patient sufficiently to enable specific anti-leukaemia therapy to be instituted. This may be by X rays, chloralkylamines 4 to 5 mgm intravenously on alternate days for 4 to 6 doses and subsequently as indicated by the white count, or Urethane B.P. 1 gm three times daily after food. When using the latter total leucocyte counts must be made every few days and treatment discontinued when the count falls to 50,000 per cu mm.

For acute leukaemia, transfusion is a temporary life saving measure, but the disease progresses to a fatal termination in a few weeks at most.

AGRANULOCYTOSIS

This is one of two grave conditions calling for urgent treatment which should be thought of when a patient presents with a sore throat (the other being diphtheria). There may be also necrotic ulceration of the mouth, pharynx, rectum or vagina and a brawny swelling of the neck. The patient looks very ill. Generalised glandular enlargement and splenomegaly rarely occur.

The onset is fulminating and commonly follows the administration of certain drugs to which the patient is sensitive. These include sulphonamides, amidopyrine, thiouracil, gold, arsenic and bismuth salts given parenterally and radio active agents.

In the circulating blood the most characteristic finding is the virtual disappearance of granular white cells (counts of 300 per cu mm are typical) while the lymphocytes remain relatively normal in appearance and number. Erythrocyte and platelet counts and the haemoglobin concentration are usually unaffected.

Treatment—This is often disappointing and the outcome largely depends on prompt diagnosis and the elimination of the causative agent. Firm handling of the situation is imperative from the moment the diagnosis is made: the patient and his attendants must be told explicitly that no medicines of any kind are to be given without the physician's consent. It must be explained that these include sleeping tablets and purgatives.

Pentnucleotide (Menley & James) 20 c cm should be given intramuscularly every four to six hours. Though intramuscular injections are painful it is best not to use the drug intravenously but it may be added to transfused blood (60 to 80 c cm to each pint).

Pyridoxine (Roche) 150 to 200 mgm intravenously is said to be a useful adjunct. Ascorbic acid 300 mgm daily should be given and ferrous sulphate gr 9 three times a day if there is anaemia.

Secondary infection of the necrotic lesions causes serious constitutional disturbances. Penicillin should be used as lozenges, sprays or powder and intramuscular injections in addition to local treatment with peroxide and saline mouth washes. *Sulphonamides must not be given to these patients under any circumstances.*

When the patient recovers he should be warned to avoid the toxic drugs already mentioned even though they may not have been definitely incriminated as the cause of the illness.

PURPURA

Treatment of patients with purpuric or other haemorrhagic manifestations depends entirely on the cause as determined by haematological examination. Thus while purpura occurs in thrombocytopenia and Hænoch-Schönlein disease, hæmophilia, hypoprothrombinæmia and scurvy it may be the first sign of

acute leukæmia or aplastic anæmia. A clear understanding of the underlying defect is therefore essential. This may be hypoprothrombinæmia, thrombocytopenia or deficiency of ascorbic acid, calcium or fibrinogen. In addition, congenital abnormalities in the capillary walls may be responsible.

Hypoprothrombinæmia with secondary purpura is caused by vitamin K deficiency. The fault may lie in the diet (low vitamin K intake), the gut (poor absorption), or the liver (inadequate conversion to prothrombin). Thus purpura of this type may be seen in obstructive jaundice, hepatic cirrhosis, and possibly following the use of certain drugs such as salicylates and dicoumarin (*see page 439*). It also occurs in pregnant women and newborn babies—*melæna neonatorum* or *hæmorrhagic disease of the newborn* (not to be confused with *hæmolytic disease of the newborn* or *erythroblastosis foetalis* which is a hæmolytic anæmia).

Treatment—Any possibly causal drugs should be stopped at once and large doses of vitamin K *analogue* (Synkavit, Roche) 10 to 20 mgm given daily by mouth, or 5 to 10 mgm intramuscularly until the prothrombin time is normal. Failing vitamin K, fresh whole blood transfusions or even intramuscular injection (as in the case of infants) may be used. Ferrous sulphate and ascorbic acid should also be given.

Thrombocytopenic purpura.

In this condition, a variable and sometimes severe degree of bleeding occurs in the form of petechiæ and ecchymoses in the mucous membranes and skin. Hæmorrhage into the brain and internal organs occurs in the severest cases. Secondary anæmia develops and is proportional to the amount of blood lost. The significant finding is a much reduced platelet count (usually below 40,000 per cu mm). In addition, a prolonged bleeding time is found but the coagulation time is normal.

Thrombocytopenic purpura may be primary (of no known cause) or secondary to the use of drugs such as gold salts, bismuth injections, arsphenamine, benzene derivatives, barbiturates, sulphonamides, thiouracil, snake venom and radium. It also occurs as a complication of infectious fevers, and as a late sign in aplastic anæmia, acute leukæmia, agranulocytosis and sometimes, pernicious anæmia.

Treatment—Since it may not be possible to decide in an emergency whether the condition is primary or secondary, any drugs which might have been responsible should be discontinued.

Fresh whole blood of appropriate group should be transfused and repeated as necessary. The object is to give red cells and hæmoglobin to relieve the severe anæmia and also to supply fresh normal platelets (which are not usually present in stored blood). Ferrous sulphate tablets B.P. gr 9 three times a day and ascorbic acid 300 mgm daily should be given.

Splenectomy is contra indicated in secondary thrombocytopenic purpura but in the primary or idiopathic type it is definitely called for at the earliest opportunity. It is important not to wait for possible recovery from the attack for the more ill the patient is the more urgent is the need for splenectomy. It should be carried out after a preliminary transfusion of fresh whole blood and in almost all cases the results are most satisfactory. Splenectomy is important because of the risk of hæmorrhage into the brain and vital organs (*e.g.* the suprarenals) which may occur without warning so long as the platelets remain low. After the operation the platelets rise steadily to abnormally high levels ultimately settling down to about 200 000 per cu mm.

Henoch Schonlein purpura

This is a type of non hereditary non thrombocytopenic purpura in which the lesions in the skin and mucous membranes are associated with urticaria joint swellings and abdominal pain and sometimes melæna. Because of pains in the limbs it is sometimes called purpura rheumatica. The disease begins in the second or third decade and continues throughout life. While it is rarely complicated by cerebral catastrophes as is thrombocytopenic purpura it often causes considerable disability.

Treatment is unsatisfactory. If the gastro intestinal tract is involved rest in bed and a light diet are indicated. For urticarial manifestations 0.5 to 1 c.c. of adrenaline solution 1 in 1 000 subcutaneously or ephedrine gr $\frac{1}{4}$ thrice daily by mouth (the last dose being given at 4 p.m.) may help. When an allergic basis can be established by skin tests or by the history desensitisation, using the appropriate solutions (supplied by Messrs Bencard and Co Gorgate Hall Dereham Norfolk) may be tried. Alternatively benadryl 100 mgm by mouth may be tried. Aspirin

should be given in the rheumatic type together with vitamin K (Synkavit Roche) 10 mgm daily by mouth Splenectomy is of no value in this type of purpura Appropriate anti anæmic treatment may be necessary

HÆMOPHILIA

In this condition bleeding may occur from many places either for no apparent reason or more often following some minor injury or operation such as tooth extraction Occasionally the presenting symptoms are caused by intra abdominal or intestinal hæmorrhage and are distinguished from those of appendicitis or duodenal perforation only with great difficulty The resulting anæmia is only slight following hæmorrhage into a joint but it may be severe after hæmorrhage elsewhere

Hæmophilia is hereditary being seen only in males but transmitted by females Hence there is often a history to aid diagnosis The significant finding on blood examination is prolonged coagulation time

Treatment—In spite of many claims there is no curative treatment but much can be done to relieve the emergencies that arise The patient should rest in bed until hæmorrhage is controlled Bleeding into a joint should be controlled by wrapping in cotton wool and immobilisation on a light splint Alternatively an ice bag may be applied to the joint but other active measures such as aspiration of blood are definitely contra indicated

Surface bleeding as from a wound or tooth socket demands local treatment Clots should be removed and small gauze dressings soaked in 1 in 10 000 solution of Russell viper venom (Boots) applied firmly In the case of a tooth socket they may be held in place by a small specially made dental splint If this fails gauze soaked in *fresh* human blood or concentrated reconstituted plasma or plasma globulin may be applied Dried plasma may be packed in a tooth socket or if available fibrin foam may be used

A newer method is to apply a 5 per cent solution of sodium alginate to the wound followed by 0.5 per cent calcium chloride solution This produces a coagulum of calcium alginate which seals the bleeding points Alginate acid powder may be dusted on a wound with similar effects

Severe bleeding calls for transfusions of whole blood. These may be alternated with transfusions of 150 to 250 c cm of liquid plasma or reconstituted desiccated plasma or plasma globulin and should be continued until the coagulation time is normal. Vigorous anti-anæmic therapy will also be required.

Emergency operations always carry grave risks in hæmophiliacs. They necessitate transfusion of whole blood preferably by the drip method before, during and after the operation.

OTHER HÆMORRHAGIC DISEASES

Purpuras caused by nutritional deficiencies

Severe purpura and anæmia are sometimes seen in scurvy. There is usually a clear history of dietary deficiency.

Treatment—The patient should rest in bed and receive a full normal diet containing plenty of protein, green vegetables and fruit and supplementary ascorbic acid 1.2 gm daily until bleeding ceases. Exceptionally whole blood transfusions may be necessary to tide over the emergency.

Bleeding caused by capillary abnormalities

In certain rare diseases such as hereditary multiple telangiectasia and pseudohæmophilia increased capillary fragility allows oozing of blood through the vessel walls. The resulting anæmia may call for emergency treatment. Local treatment of bleeding points may be needed as in hæmophilia. Good results have been claimed for Rutin—a flavonal glucoside prepared from buckwheat (Allen & Hanbury) in doses of 20 to 40 mgm three times a day by mouth.

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CHAPTER X

Fits, Faints and Unconsciousness

IT is intended here to give general directions only on how to deal with those conditions in which the urgent call is occasioned by a sudden illness in which consciousness is clouded or lost

These cases fall into two groups —

- (1) Those in which the "fit" or "faint" is of short duration
- (2) Those in which unconsciousness is profound (i.e. Coma) and relatively prolonged

FITS AND FAINTS

Almost always the attack is over or the patient obviously improving by the time the doctor arrives. Three questions should arise in his mind when called to such a case

- (1) Is it a fit (i.e., neurological in origin)?
- (2) Is it a faint (i.e. cardio vascular in origin)?
- (3) Is it some other kind of attack?

Is it a fit?

This question may be easy to decide if there is a history of previous attacks competently observed. The occurrence of clonic movements puts the attack into the category of a fit or epileptic form attack. Incontinence of urine, tongue biting, and injury in the attack also point strongly, though not conclusively to epilepsy (but do not indicate its cause). Occasionally a daytime fit may bring the doctor to a patient whose previous nocturnal attacks were unrecognised. A history of waking with a head ache and a bruised feeling in the limbs suggests convulsions during the night. Unequivocal evidence of organic nervous disease, such as cerebral tumour or general paralysis of the insane suggests that the attack was a fit.

Minor epilepsy or petit mal as a cause of "fainting" may be hard to diagnose since there are no clonic movements. Loss and recovery of consciousness are sudden, producing a mere hiatus in cerebration, post epileptic phenomena (automatism, etc.) have special diagnostic value but they are relatively rare

In a cardiovascular "faint" consciousness is more gradually lost and regained. Scars on the tongue and congenital epidermal defects suggest epilepsy.

Is it a faint?

The numerous conditions of cardiovascular origin which come under this heading may be placed in two groups

- (1) Where there is clear evidence of heart disease, e.g. heart block, aortic incompetence, etc
- (2) Where there are no signs of cardio-vascular disease, e.g. vaso-vagal attacks, postural syncope, carotid sinus syncope and transient heart block.

Is it some other kind of attack?

Two common ones should be considered

- (1) Hypoglycæmia. A clear history of insulin injected and not followed soon enough by a meal will usually point to the diagnosis but difficulties will arise if no witnesses are present. Search should be made for needle marks and a diabetic card or literature in the pockets. If in doubt dextrose should be injected intravenously. Hypoglycæmia may rarely, be spontaneous.
- (2) Internal hæmorrhage—as from a duodenal ulcer. There may be a history of dyspepsia. Increasing pallor, increasing rapidity of pulse, dyspnoea and restlessness should suggest that hæmorrhage is the cause of the faint.

COMA

Here unconsciousness is more prolonged than in a "fit" or a "faint". A classified list of causes of coma, while useful for detailed discussion is not so valuable when faced with a comatose patient as is a simple plan of enquiry. This is therefore given first.

- (1) Is there a previous history of disease which might cause coma?

e.g.

Diabetes (test the urine for diacetic acid, acetone and sugar)

Insulin Coma (If this seems likely give 20 c.c. of 25 per cent dextrose intravenously)

Nephritis (test the urine for albumin)

Pernicious malaria (examine thick and thin blood films)

- (2) Does the immediate history point to the cause?

e.g.

Injury (Do not too readily assume it is the whole cause)

Poisoning (including alcohol)

Epilepsy (Fits may be symptomatic of other disease *e.g.* meningitis)

- (3) Are there any physical signs of disease of the central nervous system?

e.g.

Cerebral vascular accidents (Lumbar puncture may be necessary)

Meningitis (Lumbar puncture is essential)

To answer these questions a full examination will have to be made and the findings recorded. This should always include the urine, the pupils and fundi, the heart rate, the blood pressure and the temperature. Lumbar puncture may be necessary.

CAUSES OF COMA

- (1) Injuries (Concussion etc. Chronic subdural hæmorrhage Electric shock)
- (2) Increased intracranial tension (Cerebral tumour)
- (3) Cerebral vascular accidents (Hæmorrhage thrombosis embolism)
- (4) Meningitis Encephalitis Cerebral Malaria
- (5) Effects of heat
- (6) Exogenous poisons (Alcohol barbiturates coal gas etc.)
- (7) Endogenous poisons (Uræmia diabetic coma cholæmia etc.)
- (8) Insulin coma
- (9) Epilepsy
- (10) Circulatory disturbances (Syncope Stokes Adams attacks)
- (11) Hysteria

C. ALLAN BIRCH

CHAPTER XI

Neurological Emergencies

THE chief neurological conditions which constitute emergencies are *unconsciousness convulsions sudden paralysis sudden blindness severe pain and vertigo*. These symptoms are not mutually exclusive but one is usually predominant. In this chapter the causes of each symptom are outlined and the underlying diseases are discussed but only *immediate* treatment is described.

UNCONSCIOUSNESS

The general problem presented by the comatose patient should be approached on the lines suggested in Chapter X.

Although the mechanism of all types of coma depends ultimately on damage to the nervous system we deal here only with the essentially neurological causes of coma which may be listed as follows —

- 1 Vascular disturbances including sinus thrombosis
- 2 Injury
- 3 Cerebral tumour
- 4 Epilepsy
- 5 Encephalitis
- 6 Meningitis
- 7 *Congestive attacks in the course of General Paralysis of the Insane*
- 8 Heatstroke
- 9 Electric shock
- 10 Hysteria

MANAGEMENT OF COMA OF NEUROLOGICAL ORIGIN — Most patients unconscious from these causes have to be removed to hospital. *Exceptions are those who have attacks of short duration such as vasomotor attacks epilepsy and proved hysteria*. It is sometimes wisest to keep a patient with spontaneous subarachnoid hæmorrhage at home at any rate at first lest his condition deteriorate under the stress of transportation. Pending the establishment of a diagnosis one must not overlook the needs of the unconscious patient (*i.e.* catheterisation and nasal feeding).

These immediate points having been covered attention can be given to the middle distance policy where the principal danger is the development of pneumonia. Aspiration of mucus from the throat should be performed hourly if necessary. nikethamide 2 to 4 c cm intramuscularly every two to four hours may aid a failing circulation.

Once general causes have been excluded examination of the nervous system should be as full as possible but may have to be modified because of the state of consciousness. Certain special devices in addition to the routine examination may be helpful in diagnosis and localisation of the lesion.

- 1 Can the patient be roused? (i.e. Is it deep sleep rather than coma?)
- 2 Note the state of the corneal reflexes and pupillary responses and whether the head and eyes are deviated to one side. (In cerebral hæmorrhage they tend to be deviated towards the side of the lesion.)
- 3 Apply painful stimuli and see if there is absence of movement in one or more of the limbs or the face. (The patient may show signs of feeling pain and yet be unable to move.)
Magnus and de Kleijn's tonic reflex is useful in diagnosis—Rotation or lateral flexion of the head to the paralysed side causes extension of the paralysed limbs in hemiplegia while movement to the normal side has the reverse effect—increased flexion and adduction of the limbs on the paralysed side.
- 4 Test the grasp reflex by drawing two fingers across the patient's palm. If positive the patient closes his hand and grasps the stimulating fingers firmly though involuntarily. When present it may indicate a lesion in the contra lateral frontal lobe.
- 5 Kernig's and Brudzinski's signs will indicate the presence of meningeal irritation.
- 6 In cases of injury comparative examination every two or three hours may be necessary to gauge progress and to discover localising signs.

THE CEREBRO VASCULAR DISTURBANCES

Sudden spontaneous happenings within the cranium are vascular in origin. Vascular accidents may be caused by—

- (a) thrombosis and embolism (*page 167*)

(b) hypertensive attacks (*page 229*)

(c) extra and sub dural hæmorrhage (*page 153*)

(d) vasospasm (*page 168*)

(e) spontaneous subarachnoid hæmorrhage This may occur at any age and result from ruptured aneurysms (congenital and acquired) tumours blood diseases or head injury Sometimes it is associated with coarctation of the aorta or subacute bacterial endocarditis The catastrophe which may be unheralded or preceded by cranial nerve affections and headache is ushered in with sudden intense headache and vomiting These symptoms and the accompanying signs arising from a combination of meningeal irritation and increased intracranial pressure tend to vary with the degree of hæmorrhage

On examination apart from coma signs of meningeal irritation are discovered The plantar reflexes are often extensor papillœdema with or without retinal hæmorrhages may be seen Pyrexia and albuminuria may be present The final diagnosis depends on the examination of the C S F which is found to be uniformly blood stained in successive samples and indeed may be so heavily contaminated as to raise doubts as to whether the theca or a vein has been punctured (*see page 377*)

Treatment—The problem is to steer the patient between the Scylla of increased intracranial tension and the Charybdis of prolongation or recurrence of bleeding The patient must be confined to bed in all cases (usually four to six weeks) If the pressure of C S F is high (over 250 mm) at the time of the first lumbar puncture it should certainly be lowered but not more than 15 c cm of fluid should be removed Further withdrawal is necessary if the blood pressure rises or other signs of cerebral compression such as deepening coma absence of corneal reflexes and fixed pupils appear

Headache should be treated by analgesics and a good combination is Heroin gr $\frac{1}{8}$ Phenacetin gr 5 Aspirin gr 5 as a powder Opium derivatives should not be used on account of their depressant effect (often fatal) on the respiratory centre

(f) Intracerebral hæmorrhage

This is most commonly caused by a rupture of an atheromatous artery in a patient who has hypertension the commonest sites being in the internal capsule (Charcot's artery) and the

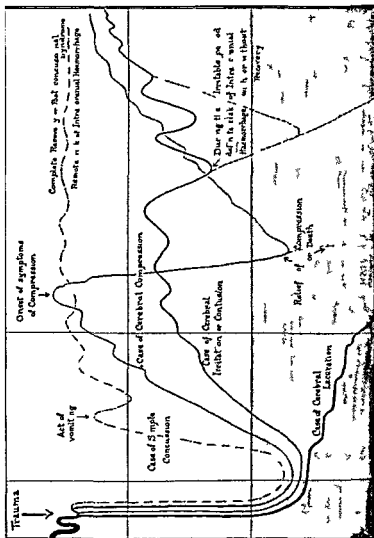


Fig 16—The effects on consciousness of a head injury

Clinical Title

The case of simple concussion

The case of cerebral irritation

The case of cerebral compression

The case of cerebral laceration

Pathological Basis

Actual laceration of cerebral tissue

By bone fragment body laceration (a) extradural (b) subdural—(1) diffuse (2) focal

General cerebral edema or cerebral contusion

No anatomical basis. Probably a physiological change

(Medical Press and Circular)

physical examination (which must include the scalp) the skull should be X-rayed. A period of observation should then follow during which the march of events is noted. Lumbar puncture and pressure reading should be delayed until the initial shock passes off, but successive punctures may give indication of the rising pressure.

CONCUSSION—As consciousness returns, sedatives should be given. During the first 24 hours, attention must be paid to the level of consciousness, pulse rate, and the development of any neurological signs. If the first 24 hours pass without evidence of complications a decision must be made as to the proposed length of stay in bed. The practitioner should sum up the case not only from the standpoint of the present crisis but also from that of the patient's general social and psychological background.

The length of post traumatic amnesia should be assessed since its duration is a rough though not infallible, guide to prognosis. Thus, mild cases of post traumatic amnesia of less than one hour should be up in three to six days, active in 10 to 14 days, and at full work within a month. In the early stages, the patient should be nursed in the position of greatest ease, and need not suffer the discomfort of being nursed flat. Gradual resumption of mental and physical activities then follows, care being taken to be neither over solicitous nor over enthusiastic in spurring the patient on.

CONTUSION—From the point of view of treatment, this may be regarded as an exaggerated form of concussion. Fluid intake should be restricted to 20 fl oz in 24 hours. Magnesium sulphate enemas (6 fl oz of 50 per cent solution) every six hours will help to reduce cerebral œdema. Only if such measures fail should reduction of intracranial pressure by lumbar puncture be used.

COMPRESSION—Surgical assistance is often required to deal with compound fractures, but in the *acute* stage help is needed only if the compression is progressive. Otherwise the general principles of management are as outlined for concussion and contusion.

CEREBRAL TUMOUR

This is usually diagnosed long before any disturbance of consciousness occurs. Occasionally, however, hæmorrhage or œdema in a tumour may cause the sudden onset of coma. In

these circumstances, the patient may be tided over the immediate emergency by the judicious use of hypertonic (50 per cent) sucrose—up to 50 c cm by slow intravenous injection, or magnesium sulphate enemata 6 fl oz of 50 per cent solution, every six hours

EPILEPSY

Sometimes it is known that an unconscious patient is an epileptic. If fits have been frequent, the doctor is likely to be summoned urgently only if unconsciousness has been unduly prolonged, and in such circumstances the possibility of barbiturate overdosage as a cause of coma in a known epileptic should be remembered

ENCEPHALITIS

The clinical picture of headache, pyrexia and disturbance of consciousness, with or without focal signs, may be produced by any of the types of encephalitis of which the following is a simple classification —

Suppurative encephalitis (brain abscess)

Non suppurative encephalitis (i) Complicating fevers

(ii) Resulting from poisons

(iii) As a primary affection of the brain

Suppurative encephalitis may present as a medical emergency when it complicates sepsis elsewhere in the body such as middle ear disease, pulmonary suppuration and pyæmia

In the acute stage extensive involvement of brain tissue may occur with diffuse cerebral softening. Sometimes extension to the ventricle occurs early, producing the picture of a virulent meningitis. When less virulent, a localised abscess forms and the clinical picture is similar to that of cerebral tumour unless and until rupture into the subarachnoid space occurs. The abscess may be extradural, sub-dural, intra cerebral or intra cerebellar so that the localising signs vary. The extra dural and sub dural abscesses tend to be complicated by venous sinus thrombosis

Treatment —Acute diffuse suppuration and rupture of a local abscess were, before the advent of penicillin, intractable conditions with an almost hopeless prognosis. Systemic administration of penicillin, 30,000 units intramuscularly every three hours, and, if necessary, intra thecal penicillin, 30,000 units daily, may offer

hope of recovery and may be combined with full sulphonamide dosage. When an abscess forms, surgical intervention is necessary.

When encephalitis is thought to be complicating an infectious fever a knowledge of the usual time of onset of encephalitis is helpful. The usual figures are —

MEASLES —Third to fifth day of illness as temperature falls and rash is beginning to fade (*page 273*)

RUBEOLA —Three to six days after the appearance of the rash

VACCINIA —Five to twenty three (usually nine to thirteen) days after the vaccination

SMALLPOX —One to twenty eight (usually eight) days after the appearance of the rash

PERTUSSIS —Two to seven weeks after onset

MUMPS —Within one week of the parotitis

In any of these diseases whenever an associated encephalitis is suspected the cerebro spinal fluid should be examined and a lymphocytosis (up to 400 per c mm) is usually found. No specific treatment is available.

Hæmorrhagic encephalitis may complicate the use of organic arsenicals. It is uncommon but has a high mortality rate. Within 24 to 48 hours of an intravenous injection of the drug headache, vomiting, convulsions and coma develop. Cranial nerve palsies are frequent. Treatment consists in repeated lumbar puncture and 50 per cent sucrose intravenously as required. Calcium thiosulphate 5 c cm of a 10 per cent solution should be given intravenously every day. The use of British Anti lewisite should be considered (*page 238*).

Other causes of encephalitis are organic solvents (*page 316*) and anæsthetics. Treatment is on general lines. Botulism is considered on *page 51*.

Virus encephalitis

All types of virus encephalitis tend to occur in epidemics and are of world wide distribution. Sporadic cases also occur. They show no apparent seasonable preference for the colder months of the year and appear to have no especially contagious characters.

SYMPTOMS —The clinical picture varies in a most bewildering fashion and is said to have undergone a definite change since the disease first came under observation. The principal acute

symptoms which may be sudden or gradual in onset are head ache disturbance of consciousness and affection of the cranial nerves. There may be inversion of the sleep rhythm with excitement whilst pyramidal and extrapyramidal manifestations with or without aphasia are common. Tendon reflexes vary but tend to be diminished. Involuntary movements of all types including hiccough may occur. Papilloedema has been noted. Cerebrospinal fluid analysis shows slight excess of lymphocytes and variable increase in the protein content.

Treatment —In the absence of specific treatment symptomatic measures are indicated. However mild the symptoms the patient should be confined to bed. Hyperexcitability demands the use of sedatives *e.g.* hyoscine hydrobromide gr $\frac{1}{100}$ to $\frac{1}{200}$ six hourly for two days supplemented if necessary by phenobarbitone gr $\frac{1}{2}$ or more. If comatose the patient should be dealt with on the general lines already indicated.

MENINGITIS

All types of meningitis present a number of symptoms and signs in common. Severe headache is usual but the seriousness of the illness is more often proclaimed by clouding of consciousness. Meningitis is therefore considered here under the heading of coma. Diagnosis is rarely difficult. Headache increases in severity and in severe paroxysms it may radiate to the neck and cause the patient to scream in anguish. The meningitic syndrome completes the diagnostic picture the victim is curled up in an attitude of general flexion resentful of interference suffering from photophobia and in children especially vomiting and convulsions punctuate the progressive deterioration in the general condition.

Physical examination confirms the diagnosis and the signs can be explained on the basis of increased cerebrospinal fluid pressure (causing retinal congestion and choked optic discs) and meningeal irritation which produces the classical signs of Kernig, Brudzinski and Guillain. If unrelieved the increased intracranial pressure is inevitably reflected in depression of vital functions at various levels. Slow and sighing respiration and interference with sphincter control are heralds of impending death. Pupillary abnormalities and disturbances of superficial reflexes are rarely absent but vary

considerably from patient to patient and in the same subject from hour to hour

Given this combination of symptoms and signs the diagnosis of leptomeningitis is almost inescapable but we must remember that other pathological processes may produce a similar clinical picture These are —

- 1 *Acute general infections* such as pneumonia These may produce headache and some of the signs (including an extensor plantar reflex) but not the serological evidence of meningitis (Meningism)
- 2 *Incipitilis* may cause signs of meningeal irritation
- 3 *Subarachnoid hæmorrhage* closely simulates meningitis and in young persons with normal arterial systems may be clinically indistinguishable

THE PRECISE DIAGNOSIS —The establishment of the diagnosis of leptomeningitis as opposed to the three conditions listed above and the determination of the underlying cause upon which therapy and prognosis depend can be achieved only by examination of the cerebro spinal fluid Nevertheless certain clinical features may aid in the process of differentiation and accordingly we shall mention some of these ancillary signs and briefly discuss their significance

The common organisms causing meningitis are the meningococcus the pyogenic organisms such as streptococci staphylococci and pneumococci the tubercle bacillus hæmophilus influenzae the virus of lymphocytic chorio meningitis and other viruses and the spirochæta pallida

Although identification of the causal organism can only be made by examination of the C S F certain clinical findings may point to the probable diagnosis Thus meningococcal infection is suggested by a petechial rash and the presence of other cases and also in rare cases by severe collapse and shock (Waterhouse Friderichsen syndrome 119 225) A pre existing focus of infection such as otitis or pneumonia suggests a pyogenic meningitis Fundal hæmorrhages may occur in subarachnoid hæmorrhage An insidious onset and a history of contact with an open case of tuberculosis is commonly found in tuberculous meningitis

Meningitis is a rare complication of secondary syphilis Its onset may be insidious with symptoms very similar to those of the meningo vascular type of tertiary neurosyphilis

Occasionally the picture is that of an acute meningoencephalitis attended by convulsions coma and the signs of an acute meningitis. A similar picture may also occur as an episode in the tertiary stage. As in the other types of meningitis the diagnosis is established by the examination of the cerebrospinal fluid which shows a lymphocytosis up to 1 000 cells per c mm and a positive Wassermann reaction.

INTERPRETATION OF CEREBROSPINAL FLUID FINDINGS—As soon as practicable lumbar puncture must be performed. Inspection may reveal opalescence or turbidity thus establishing the existence of a pyogenic process although in very early stages this may not be apparent to the naked eye. Specimens of fluid totalling 8 c cm should be sent at once to the laboratory for estimation of proteins sugar chlorides cell count Wassermann reaction Lange curve and above all culture for the identification of the organism.

Treatment

MENINGOCOCCAL MENINGITIS—Before the advent of sulphonamides meningococcal meningitis carried a high mortality (65 to 75 per cent) and among survivors neurological sequelæ were often extremely disabling. With modern therapy the disease can be classed among the infections of moderate severity and the mortality has fallen to about 5 per cent. The drug of choice in the present state of our knowledge is sulphamezathine and should be given in doses according to the table on page 440 if this is not available use sulphadiazine sulphathiazole or sulphapyridine in this order of preference. Penicillin which must be given intrathecally though effective is unnecessary. Intrathecal injection of sulphonamides is strictly contra indicated in any form of meningitis. (*For treatment of the Waterhouse Friderichsen syndrome see page 225*.)

PNEUMOCOCCAL STREPTOCOCCAL AND STAPHYLOCOCCAL MENINGITIS—The application of penicillin in the treatment of these infections of the meninges has opened a new field in therapeutics. The final decision has not yet been made on the precise regime to be adopted but satisfactory results have been obtained by the administration of penicillin alone using a combination of intrathecal and intramuscular therapy. On the whole however it would appear wise to use both penicillin and sulphonamides. Care should be taken to remove any focus of infection particular attention being paid to the presence of otitis.

The penicillin and sulphonamide (sulphadiazine) therapy should run concurrently (*For dosage see page 440*)

HÆMOPHILUS INFLUENZÆ MENINGITIS—Early reports suggested that hæmophilus influenza was not susceptible to penicillin but more recently it has been shown that certain strains respond to penicillin and to sulphonamides. In the circumstances therefore it is advisable to try the combined therapy as used for pneumococcal meningitis if necessary increasing the dose of intramuscular penicillin up to 50 000 units every three hours. Rabbit serum has been tried with success (*see page 275*)

ACUTE LAMINOCTIC CHORIO MENINGITIS—This disease is self limited and clears up in about fourteen days. No special therapy is required. The prognosis is excellent.

SYPHILITIC MENINGITIS—This meningitis acutissima has previously been treated by various intensive courses of arsenic and bismuth with varying success. Its relative rarity makes therapeutic trials necessarily restricted. A priori evidence however suggests that acute syphilitic meningitis should be treated by a full course of intra thecal and intramuscular penicillin as outlined for pneumococcal meningitis but continuing the intramuscular course for up to 21 days. Subsequently routine arsenical and bismuth therapy should be instituted.

CONGESTIVE ATTACKS OF G P I

These resemble cerebral hæmorrhage in their acute onset with interruption of consciousness varying from drowsiness and confusion to complete coma. No special emergency treatment is indicated.

HEATSTROKE (*See page 305*)

ELECTRIC SHOCK (*See page 88*)

HYSTERICAL UNCONSCIOUSNESS

True unconsciousness does not result from hysterical causes. If a patient is genuinely comatose organic disease must be assumed to be present even if there is a history of hysterical manifestations.

If hysteria is suspected an endeavour should be made to establish a definite diagnosis by showing a source of conflict. A full physical examination should be made. Normal ocular and tendon reflexes are strongly suggestive that there is no underlying organic disease. So also is the absence of stertorous breathing. If there is paralysis it is usually bizarre and any convulsive

movements are irregular and do not follow the tonic clonic succession of epilepsy. Flickering movements of the eyelids are commonly seen in these cases. In hysterical pseudo coma attempts to open the eyes are resisted by the patient and pressure on the supra orbital nerves or temporary obstruction of the nose and mouth will rapidly rouse the patient.

CONVULSIONS

Convulsions always present to the practitioner a serious problem in diagnosis and treatment. The occurrence of a fit especially for the first time strikes terror into the mind of the layman, impels the immediate attendance of the doctor and may betoken the onset of a serious disease requiring urgent treatment.

The functional unit consisting of upper motor neurone, lower motor neurone and muscle can react to insult in only two ways. It may become paralysed or it may produce an abnormal contraction of the muscle. A repetition of muscular contractions attributable to irritation of the upper motor neurones constitutes the convulsion of clinical medicine. It is not surprising therefore to find an imposing list of causes of convulsions and it is helpful from the point of view of diagnosis to group these causes according to the age at which they occur.

Convulsions in children are dealt with on page 262. Here we shall deal with *convulsions in adults*. The immediate problem may be to deal with the attack. If seen early enough, i.e. before the jaws are in spasm, dentures should be removed and a handkerchief or soft gag should be inserted between the jaws. Spectacles should be removed and the collar loosened. Beyond preventing the patient from hurting himself, no restraint should be imposed. If no underlying disease is suspected, the patient may resume normal activities after 15 to 30 minutes' rest, but many will sleep for a longer period.

If the whole attack is witnessed, special points to observe are the origin and spread of movements, the development if any of unconsciousness, and the occurrence of incontinence, tongue biting and other injury. More often the course of the attack is not observed and we have to rely on the evidence of a witness who is usually unreliable and often obtuse.

Enquiries and examination should be directed to settling the following points —

- (1) Is the attack of psychological or organic origin?
- (2) If organic in origin is it attributable to neurological or general disease?
- (3) If neurological is it a recurrent disturbance of function of idiopathic type or is it a manifestation of a progressive process?

A safe guiding principle is that all convulsive phenomena in adults are caused by progressive pathology *until proved otherwise*. Questions should be directed both to the patient (if able to respond) and to an eye witness and should aim at obtaining as complete a picture as possible of the attack with its prodromal and post ictal phases. Answers should be sought to the following questions—

- (a) Is this the first attack?—if not when did they begin and how often do they occur?
- (b) Are the convulsions generalised from the outset or do they begin in one particular area and spread?
- (c) Are they accompanied by unconsciousness preceded by any special aura or followed by any particular abnormalities mental or physical?
- (d) Has the patient been complaining of symptoms of general disease recently or did the convulsion occur unexpectedly?
- (e) Have there been any symptoms to suggest that the convulsive attack is simply an episode in a progressive neurological disorder such as tumour arterial disease or luetic affections? If a female is she pregnant?
- (f) Is there a family history of fits?
- (g) Has the patient suffered any recent injury which might produce either cortical scarring or tetanus?
- (h) Is there any evidence of intoxication?

In carrying out the physical examination after the attack it should be remembered that most patients are unconscious for at least a few minutes after the attack although the focal type of attack may not be accompanied by any loss of consciousness.

Any difference in reflexes motor power and sensation on the two sides of the body should be noted. Both plantar responses are commonly extensor for a brief period after the attack and any difference in the two sides may suggest the presence of a focal lesion. Focal attacks are frequently followed by weakness of the parts affected (Todd's palsy). With organic

usually the signal for the development of acute anxiety in all concerned. Fear of its immediate and long term effects coupled with the publicity given to the disease in this country and America have naturally induced emotional attitudes which do not aid in management. The situation has been further complicated by the controversial evidence as to the mode of infection and portal of entry. In the light of present knowledge it is safe to assume that the disease may enter via the nasopharyngeal and gastro intestinal routes and therefore the recommendations we make are as follows.—

- 1 Institute complete barrier nursing for 28 days
- 2 Spray the room with D D T to prevent insect contamination
- 3 Dispose of faeces without danger of contamination (page 280)
- 4 Isolate child contacts for 21 days (The estimated period of incubation is 12 days) It must be remembered that persons capable of transmitting the disease may never show signs of infection themselves. If the outbreak occurs at a residential school or other institution it is not advisable to send the children home since this opens the possibility of spread to younger and more susceptible children. If a case occurs in a residential hospital where the contacts are adults it is felt that no isolation of contacts is necessary.
- 5 Consider the use of immune serum in epidemics for the protection of child contacts. Convalescent serum 10 to 20 c cm or immune horse serum 10 c cm may be used. Experimental work on active immunisation and nasal spraying has provided no evidence in favour of these methods of prophylaxis.

CARE OF THE PATIENT—In the acute phase i.e. before paralysis general nursing attention will be required. Analgesics should be given for pain. Gentle passive movements of the limbs are helpful and hypertonic saline baths are soothing for hyperæsthetic limbs. Foot boards should be applied to prevent early footdrop. The cabinet respirator should be made ready (page 407).

If paralysis has occurred the part affected should be immobilised in a position which will avoid over stretching of the paralysed muscles. Muscles to which special attention should be given are the deltoids the small muscles of the hands the rotators of the hip the extensors of the hip and knee and the dorsiflexors of the foot.

Acute infective polyneuritis

In this condition a short period of pyrexia is followed by a spreading flaccid muscular paralysis pains and paræsthesiæ. Examination reveals tender muscles diminished reflexes and hyperæsthesia or anæsthesia of glove and stocking type. The cerebro-spinal fluid shows a raised protein content (100 to 400 mgm per cent) with little change in cell content (dissociation albumino cytologique).

Involvement of the vagus may cause troublesome tachycardia. Respiratory paralysis may also be an urgent symptom and call for the use of the respirator. Recovery is the rule but occasionally a patient is seen whose condition deteriorates steadily until death occurs from respiratory failure.

Myasthenia gravis

A case of myasthenia gravis (often misdiagnosed as psychoneurosis) may be seen for the first time in a crisis with cardiac and respiratory failure imminent.

The myasthenic face (drooping jaw bilateral ptosis and wrinkled forehead) together with a history of being easily fatigued should suggest the diagnosis. Prostigmin 2.5 mgm with atropine gr $\frac{1}{100}$ should be given subcutaneously at once and repeated as necessary.

Artificial respiration and emergency treatment for pneumonia may be needed together with nasal and parenteral feeding. After the immediate crisis is over prostigmin can be given orally—15 mgm three four or more times daily.

Familial periodic paralysis

Like myasthenia gravis this rare disease is likely to be mistaken for hysteria or a psychoneurosis. Its pathogenesis is obscure but it is thought to originate in a disorder of sodium and potassium metabolism. The victim of this remarkable disease suffers periodically from bouts of flaccid paralysis lasting from three hours to four or five days. Its onset is characteristically in the teens but occasionally it occurs in early childhood.

Paralysis usually occurs soon after the patient awakens and may have been preceded by stiffness hunger and thirst or other prodromata on the previous day. Precipitating causes are cold starvation alcohol fatigue and menstruation. The paralysis

reaches its peak in one or two hours and is usually symmetrical but may be of hemiplegic or paraplegic distribution and occasionally the respiratory muscles are involved

The history of previous attacks and the familial evidence should point to the diagnosis. Examination reveals a flaccid paralysis with diminished reflexes in contrast to hysteria where they tend to be brisk. Two other features help to differentiate the condition from hysteria: the blood potassium is low (14 mgm per cent) and the muscular responses to galvanism and faradism are diminished or lost.

Treatment—Potassium chloride 12 to 16 gm should be given by mouth and repeated if necessary. A respirator may be needed.

EMERGENCY TREATMENT OF PARAPLEGIC CONDITIONS

Having made a diagnosis of paraplegia or hemiplegia for which there is no specific treatment it is not sufficient to rest content with general nursing care. The prognosis depends on proper management as soon as the emergency state begins.

Some of the factors determining the outcome such as the extent of the initial motor weakness and sphincter disturbances are not influenced by treatment. Urinary and pulmonary infections, pressure sores, contractures and the extent of residual loss of function can undoubtedly be influenced by correct treatment.

Sphincter disturbances

Retention of urine is the commonest sphincter disturbance calling for urgent treatment.

- 1 If retention results from *tabes dorsalis* or is of recent origin and unaccompanied by widespread neurological signs, carbachol B.P. 1 c cm intramuscularly should be used. (Beware of over dosage and never use it except in solutions from an ampoule. It must never be given intravenously.)
- 2 If retention is part of the picture of coma, repeated catheterisation is the best course.
- 3 Retention complicating serious disease of the cord such as compression or inflammatory or degenerative processes demands prompt treatment. Slow decompression by catheter is the first step followed by the establishment of tidal drainage (page 386). Suprapubic drainage has no place as an immediate measure.

Retro bulbar neuritis

This is characterised by sudden loss of vision usually in one eye accompanied by aching pain in the orbit. On examination a central scotoma can be found. The pupil reacts sluggishly to light but the consensual reaction is brisk.

The treatment is that of the causal condition and may as in the case of acute sinusitis and methylated spirit poisoning be urgent (*see page 10*).

Hysterical blindness

This may be unilateral or bilateral partial or complete. The patient avoids obstacles in his path and shows the blink reaction to menace. On examination the pupils and fundi are normal. Unilateral hysterical blindness may be detected by the use of red green glasses combined with a word test using red and green letters.

PAIN AS A NEUROLOGICAL EMERGENCY

Four questions should be asked about any pain —

Where is it? (Site and radiation)

What is it like? (Character and intensity)

What makes it worse?

What makes it better?

While we are here discussing only those diseases classified as neurological it must be borne in mind that in practice this arbitrary grouping does not exist and that severe pain may be other than of neurological origin. Even patients with known nervous disease may have appendicitis and gastric ulcers are commoner than gastric crises.

Headache

The degree of anxiety and consequent apparent urgency caused by any painful condition varies with the patient's psychological make up. We must not be misled by the impassiveness of the phlegmatic patient on the one hand or the anxiety of the nervous patient on the other remembering that the significance of a pain varies inversely as the richness of its description. This is particularly important in evaluating the significance of headache.

Severe headache presenting as an emergency may be a symptom of the following conditions —

- (1) ANXIETY AND HISTERIA —Headache in these conditions is often described as "terrible" or "agonising" but is usually qualified by the terms "pressure" or "bursting". The immediate point in management is to exclude organic causes and then to approach the problem confidently as a psychoneurosis. The ophthalmoscope should always be used.
- (2) SUBARACHNOID HÆMORRHAGE —(See page 150)
- (3) MENINGITIS —(See pages 159 and 274)
- (4) INCREASED INTRA CRANIAL PRESSURE FROM ANY CAUSE
- (5) MIGRAINE —The headache of migraine, especially when it occurs for the first time, may produce terrifying effects in the patient and call for judgment in diagnosis and treatment. Characteristically the attack begins with a visual aura, black spots before the eyes, hemianopia (to the patient's great distress at first) and fortification spectra. There may be a sensory aura of tingling in the limbs or face. Not infrequently weakness of the limbs also occurs. After ten to twenty minutes the crushing headache develops, usually unilateral, but occasionally of general distribution. Nausea and vomiting may follow.

The duration of the attack may be from one hour to an entire day. On examination the hemianopia may be detected and an associated spasm of the retinal arteries seen. The nature of the condition is probably a vascular spasm followed by dilatation, but it should be remembered that similar symptoms may result from aneurysm, hypertension and uraemia. Ophthalmoplegia may occur in true migraine but should raise the possibility of aneurysm or tumour.

Treatment —Once the attack is well under way, little can be done, but certain drugs may be of value in the early stages. The following are alternatives —

- (a) Ergotamine tartrate (Femergin) 1 mgm sublingually, followed by 1 mgm in one hour. It should not be given to pregnant women.
 - (b) Femergin, 1 mgm subcutaneously.
 - (c) Benadryl, 100 mgm followed by 50 mgm in two hours.
- The patient should be advised to rest.
- (d) Adrenaline solution 5 to 10 minims subcutaneously.

Pain in the trunk (root pains).

Not infrequently one sees in the neurological out-patient clinic, a patient in such acute agony that he beseeches the doctor to give him relief, and threatens suicide if nothing is done to abolish his pain. These unfortunate people present problems both of immediate therapy and long term diagnosis. Careful questioning elicits the fact that the distribution of the pain corresponds anatomically to root distribution and is usually accentuated by coughing and sneezing. The possible causes are tabes dorsalis, secondary carcinomatous deposits, extramedullary spinal tumours, Pott's disease, spondylitis, meningeal inflammation, herpes zoster—in the pre-eruptive stage—and hysteria.

The characteristics of the pain should indicate the diagnosis, but the following points may be useful—

- (1) Girdle sensations of tabes dorsalis will be accompanied by corroborative signs in the pupils and tendon reflexes.
- (2) Severe root pains in an elderly person, in the absence of signs of syphilis, are usually caused by secondary neoplastic deposits.
- (3) The presence of kyphosis suggests tuberculous disease of the spine.
- (4) If the location of pain and altered sensibility do not coincide with root distribution, the condition is either attributable to general disease or to hysteria. Visceral referred pain often assumes a root distribution and must be considered.
- (5) It must not be forgotten that patients with neurological disease sometimes have appendicitis or perforated ulcer.

Treatment—Having excluded a surgical cause it is usually necessary to give symptomatic treatment. Whilst realising the dangers of addiction we feel that too often a patient is allowed to suffer untold agonies for the want of adequate analgesia. In painful incurable disease with a short prognosis—such as cancer with metastases—the correct dose of analgesic is *that which relieves the pain*. Morphine is ideal, but another useful preparation is—

R. Heroin	gr $\frac{1}{8}$	
Aspirin	gr 5	Make a powder
Phenacetin	gr 5	

One powder every four hours as required

For the tabetic little can be done though Aspirin gr 5 and

Codeine gr $\frac{1}{2}$ may give relief. Hysterical pain should not be treated regularly with analgesics liable to cause addiction.

Visceral pain of neurological origin

Acute abdominal pain with vomiting, spasm of the larynx, bladder and rectum may occur as part of the picture of tabes. No doubt about the diagnosis need occur if reasonable powers of observation are exercised and examination of pupils and tendon jerks is carried out.

Treatment—For relieving laryngeal crises, amyl nitrite is usually satisfactory. Gastric crises are helped by morphine but again there is the danger of addiction. Intramuscular sodium phenobarbitone gr 3 repeated after four hours if necessary will help to relieve pain and promote sleep.

Other types of pain

Peripheral nerve disease including sciatica, spinal cord degeneration and neoplasia and affections of the thalamus may at times produce very severe pain for which adequate analgesics should be prescribed pending diagnosis. As alternatives to morphine and heroin the following prescriptions are helpful—

1	Phenacetin	gr 5	As a powder every four hours
	Aspirin	gr 5	
	Codeine	gr $\frac{1}{2}$	
2	Sodium phenobarb	gr $\frac{3}{4}$	One ounce four times a day
	Sodium bromide	gr 10	
	Phenazone	gr 15	
	Tincture of Gelsemium	m 20	
	Chloroform water	fl oz 1	
3	Codeine	mgm 30	Every four hours
	Pethidine	mgm 50	

VERTIGO

Vertigo may sometimes produce terrifying effects on the patient and in this section only the severe and urgent cases are considered. They may be caused by—

- 1 **Menière's Syndrome** This consists of paroxysmal vertigo associated with deafness and often with nausea, vomiting and loss of consciousness. The condition has a variety of causes but is essentially a disturbance of the labyrinth. Paroxysms are recurrent and deafness progressive.

- 2 Otosclerosis and Eustachian tube obstruction
- 3 Migraine
- 4 Acute suppurative ear disease with or without intracranial (cerebellar) extension
- 5 Brain stem affections particularly disseminated sclerosis and posterior inferior cerebellar artery thrombosis. The associated signs confirm the origin of the vertigo. In posterior inferior cerebellar artery thrombosis infarction of a wedge shaped area on the side of the medulla causes a characteristic syndrome with severe vertigo and some inco-ordination on the side of the lesion. On examination there is Horner's syndrome (miosis, enophthalmos and ptosis from paralysis of the ocular sympathetic) and paralysis of the palate, pharynx and vocal cord all on the side of the lesion. Analgesia and therm anaesthesia are found ipsi lateral for the face and contra lateral for the limbs and trunk.
- 6 Sudden haemorrhage particularly in the region of the cerebellum and usually from an aneurysm may result in vertigo.
- 7 Epilepsy. Here vertigo may be part of the aura.

MANAGEMENT—No matter what the cause of the vertigo the patient should be confined to bed. If intracranial abscess is suspected removal to hospital is indicated. For the vertigo of Ménière's syndrome and migraine morphine gr $\frac{1}{4}$ and hyoscine gr $\frac{1}{200}$ is very helpful but even chloretone gr 20 may be adequate. Benadryl 50 to 100 mgm may be tried whilst welcome sleep and symptomatic relief may result from sodium phenobarbitone gr 3 intramuscularly. These remedies may also be of service in alleviating the vertigo of posterior inferior cerebellar artery thrombosis and disseminated sclerosis though caution is required in vascular conditions lest the cerebral depression resulting from the therapy should prejudice ultimate recovery. It is better therefore to use chloretone gr 10 or small doses of sodium phenobarbitone (1 to 2 gr intramuscularly) in such cases.

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CHAPTER XII

Psychiatric Emergencies

(For psychiatric emergencies on board ship
see page 343)

PSYCHIATRIC emergencies are those in which the behaviour of the patient is socially disturbing. They may arise because the patient —

- (1) is excited restless and impulsive
- (2) is agitated self accusatory and suicidal
- (3) is in a state of terror and panic
- (4) is acutely enraged and destructive
- (5) is resistive or semi stuporose
- (6) is confused and suffering from false sense impressions (hallucinations) or false ideas (delusions)
- (7) is having hysterical fits or after epileptic attacks is behaving in anti social ways
- (8) has attacks of powerlessness or severe pain
- (9) has a prolonged loss of memory or a fugue

This classification however obscures one important fact. A state of emergency exists in many instances long before exuberant symptoms appear. This is particularly so in the deliria *e.g.* alcoholic delirium where the early slight confusion and irrelevant talk and fine tremor constitute danger signals possibly days before classical delirium tremens appears. In the same way nocturnal wanderings in the elderly or excessive irritability in the epileptic should call for careful evaluation.

Now whilst diagnosis is essential ultimately the state of emergency necessitates our following general rules first. To do so we may emphasise two points. First even the acutest emergencies *may* be transitory. However florid grotesque and socially disrupting the patient's behaviour it may be very short lived. But the illness can only be short lived if the correct treatment is given and that may entail removal not only from home but to a mental hospital. Sudden psychotic behaviour by no means spells chronic mental illness. We are justified therefore in maintaining a hopeful attitude providing we can ensure the relatives

co operation. But we cannot be hopeful unless they will implement our plans, however distasteful. Secondly, the patient's behaviour will have created dismay, if nothing worse, and therefore uncertainty, in his mind. One of our tasks must be to try to overcome this. Where it is possible, see him alone. We may find that he is anxious for more help from us than we expected. In a small number of acute cases, complete misunderstanding is at the bottom of bizarre behaviour. Be on the look out for such cases. To be sympathetic one must also be firm. Where unwelcome facts dominate the picture be prepared to state them. The subsequent course of a mental breakdown is often determined by the wise handling of it or otherwise, in its critical early stages.

We may now divide emergencies into (1) those where we can approach the patient and discuss his illness, and (2) those where approach and discussion are impossible. In both types physical examination must be made. This must include more than a cursory examination of the chest and abdomen. Is the patient anæmic or ill nourished? Is vitamin deficiency evident? Examination of the pupils, of the tendon jerks of the plantar response and of the fundi (when possible) should all be included.

Despite the patent mental abnormality, the physical factor may be primary. This is especially true of the confusional and delirious states, *e.g.* alcoholism, vitamin deficiency, bromism, cerebral abscess, and secondary carcinoma of the brain. Whilst the anxious practitioner may feel that immediate transfer elsewhere is the wisest course, the rash doctor may keep his patient at home too long. Adequate treatment of the first three examples at home is most rewarding, but is clearly unwise in cerebral abscess. The facts and dangers as well as the hopes must be faced candidly with the relatives. Serious social disturbance, such as suicidal gestures, may be temporarily controlled with heavy sedation, whilst the measures appropriate for the underlying physical disease are taken. But refusal of food and serious under nutrition occurring in a confusional state call for transfer at once to a suitable hospital or nursing home.

The obverse picture of physical symptoms occurring in what is primarily a psychiatric condition are frequently met with as manifestations of an hysterical personality. They are rarely emergency situations but they sometimes become so, as in

patients with visceral pain who are admitted to hospital as emergencies. The writer has the impression that practitioners sending such cases to hospital are perhaps loath to divulge the fact that the patients have shown hysterical tendencies previously but they would be well advised to do so.

In epileptic attacks a somewhat similar emphasis upon the physical may cause one to overlook the psychiatric features of the case. Is the post epileptic confusion of the usual short lived type or are there features which should make one specially investigate the possibilities of G P I or an epileptic dementia with loss of judgment and anti social acts?

Though presence of physical disorder may not alter the immediate treatment its possible presence must come into our mind. Further faced with the emergency everything will be gained if we can indicate our concern and yet our belief that we can help. We may be able to communicate to the patient the fact that misunderstanding and exhaustion has precipitated his illness. We must convey the fact that we intend willy nilly to direct his treatment or disposal. Every patient who becomes a psychiatric emergency is exhausted whether by fever toxins endocrine imbalance emotional conflict or most common of all by his relatives well meant but ill advised solicitations. The rapidity with which the hysteric's outbursts or fits may subside is one example of what results from an altered environment whether in hospital or by the exclusion of relatives from his room at home. All doctors have seen the marked change in behaviour which follows the installation of a well trained nurse.

DRUGS

Although nursing and the attitude of the environment—firmness and informed hope superseding capricious handling and alarm—may be said to be as important in the long run as the exhibition of drugs these may be necessary in the acute stages when all the above principles may break down.

Morphine up to gr $\frac{1}{2}$ with hyoscine gr $\frac{1}{15}$ may be needed and may have to be injected by force the police having been called in. This method or anæsthetising the unwilling patient may have to be resorted to in order to save himself or others. Such measures however should be the last resource rather than the first.

We frequently need time to think and to discuss ways and means. In these circumstances intravenous barbiturate injection is the method of choice [0.75 gm thiopentone (pentothal) mixed with 10 c cm sterile water which should be slowly injected]. The whole position may be so difficult that these measures are felt to be too transient and then paraldehyde is the drug of choice. Paraldehyde in the writer's opinion should be more readily available than it is. A small bottle (4 fl oz) containing paraldehyde m 120 compound tragacanth powder gr 10 water to 1 fl oz may be carried though it must be renewed every month. Paraldehyde may be given by injection into the glutei in m 180 doses (about 10 c cm). Preferably it is given by mouth if the patient will take it (e.g. in deliria m 180 by the mouth and repeated in two hours or longer) or per rectum if a nurse is available. The dose per rectum should be $\frac{1}{2}$ fl oz mixed with 10 fl oz of a 5 per cent solution of glucose. Glucose is preferable to saline in preventing proctitis.

Is the patient fit to remain at home? Are special nurses needed?

As indicated above the answers to these questions depend partly upon the type of disturbance met with the degree to which drugs can be expected to control the disorder and the number of suitable nurses who can take charge. If there is any doubt about the two latter considerations removal to hospital is essential.

Acute excitement may be checked temporarily by large doses of sedatives and provided the risks are understood the relatives' plea to keep the patient at home may be accepted. The real danger however is that drugs in the necessary amounts are themselves toxic and removal from home when they have only modified the excitement is a more dangerous physical risk than at the earlier stage. Suicidal gestures must be prevented. It is not sufficient to accept the relatives' promise that the patient will be watched. Relatives are not trained and they do not know all the risks. To inform them usually makes them even more harassed. Only well trained nurses are competent and it may be necessary to employ two by day and two by night. Very few persons can meet such a financial strain. Special nurses however well trained can be good or bad. They will always be bad if

they cannot compete with the relatives. In such cases do not be led into temptation. Insist upon removal from home.

It cannot be too emphatically stated that malnutrition in the psychotic is far more dangerous to life than in the physically ill. The patient who does not take sufficient fluid and solid nourishment must be removed unless a nurse can take charge and ensure adequate fluid intake at once.

There is another aspect of the answer to the question we have formulated. Although excitement, suicidal risk, and refusal of food are the clinically clamant features, the importance of routine in the nursing of psychiatric cases is very great. This is observed in chronic cases where dramatic social improvement is often seen on removal to hospital. It is equally important in acute cases. Some relatives respond at once and give the special nurse and doctor every support. Such instances are not the commonest and unhurried sympathetic firmness is called for. These principles also apply to certain cases of hysterical personality whose socially disturbing conduct is likely to continue until they realise that the medical man insists on removal to another environment.

We are now in a position to deal with the particular types of psychiatric emergency.

STATES OF EXCITEMENT

In extreme cases the patient is restless, interfering, over talkative, sleepless and too absorbed in his fanciful world to take regular meals or stuffs the food in when he eats at all. Rapid sedation is essential. If paraldehyde is refused, morphine gr $\frac{1}{4}$ to $\frac{1}{2}$ with hyoscine gr $\frac{1}{100}$ will be necessary for an intravenous injection of barbiturate will be impracticable. Transfer to a mental hospital is essential. The doctor is called in early in such cases and as worse symptoms are likely to develop, no compromise of a temporising kind should be made.

In less severe cases the temptation is to give sedatives (barbiturates are the best, e.g. phenobarbitone gr 2 and then gr 1 three times a day). If doses of this degree are required, there is a clear indication for transfer to a suitable hospital. Phenobarbitone should not be continued in this amount to patients without nursing supervision.

STATES OF AGITATION

The patient is exceedingly restless, usually talks on one subject—his approaching doom, or that of his near ones, is filled with remorse, and his groans and cries as well as his importunity are socially disturbing. Such patients may wrongly be dismissed as "hysterical" if it is thought that they are exhibiting self-pity. The danger of suicide cannot be brushed aside. When in doubt, remove the patient to a suitable hospital. Undoubtedly some patients' agitation has been over-valued, and much criticism has been made of the doctor who has caused them to be placed under restraint. Although this may appear regrettable a patient who has apparently overplayed his wories is not as seriously handicapped by such removal as is supposed. He cannot remain "certified" and the precautionary nature to him of such treatment may have beneficial effects awakening his lost sense of responsibility towards others.

When less risk is present sodium amytal in capsules of gr 3, three or four times a day is useful, or tincture of opium, m 10, liquid extract of cascara sagrada m 10, chloroform water to $\frac{1}{2}$ fl oz should be prescribed three or four times a day, and specialist advice sought.

TERROR AND PANIC

An unreasoning dread of impending disaster dominates the picture, which is acute or sub acute in onset, and may result in a mixture of excitement and agitation (rushing hither and thither, complete obliviousness to others' fears, frantic planning followed by vacillation and uncertainty, indifference to dangers, resulting in unpremeditated exposure to risks of death or stuporose withdrawal and a facies betraying extreme fear).

Panic is transitory and calls for immediate sedation. Thio-pentone (pentothal) anæsthesia is the method of choice. On regaining consciousness the patient is quieter and can discuss the precipitating factors. Arrangements must be made for the elucidation of his terror state, and for the proper treatment of the factors internal (in the patient's make-up) and external (in his circumstances).

PHOBIAS

These are less severe states than panic, recurring in special conditions (open spaces, crowded rooms, closed vehicles, or in

the presence of certain animals etc) They rarely merit description as psychiatric emergencies except when occurring in hysterical personalities or in children at night In the latter an element of mental confusion may be present and parental anxiety may be extreme and a great handicap for the sense of security which the child requires is conspicuously absent The doctor's task is reassurance of the adults and the inculcation of methods of wiser handling The services of a Child Guidance Clinic or psychologically minded pediatrician should be sought A quickly acting barbiturate e g sodium amytal gr 3 should be given

RAGE AND DESTRUCTIVENESS

These are commonly called brain storms and may indicate the onset of a severe psychosis They more frequently indicate a severe hysterical reaction in a poor personality with or without mental or physical handicaps Like hysterical fits they may seem to be purposive and are always dramatic Immediate removal of all outside spectators including the family is called for and sympathetic firmness from the doctor who remains to hear the patient's story Such states are associated with extreme exhaustion consequent in part upon depletion of the glycogen reserves Small doses of a barbiturate (sodium amytal gr 1) and easily ingested sugar should be given More insight into the condition is required and is better dealt with the next day and without fuss rather than after a long interval If a long period intervenes the patient will have become more dependent upon the recurrent excitement his behaviour promotes If dealt with early a much more (apparently) off hand ready readjustment is possible

STATES OF STUPOR AND RESTIVENESS

All degrees of slowing of thought and action from simple retardation to extreme stupor may be met with and are usually associated with restiveness Because the condition is not socially disturbing no advice may be sought until the patient is speechless and foodless A similar state may occur after an infective illness and what was thought to be convalescent weakness proves to be a state of stupor In either case the dangers associated with a severe toxic condition are very real Skilled treatment is urgently indicated

Although much may be done with adequate nursing at home diet and vitamin therapy the underlying condition is seldom relieved and these conditions may so rapidly progress to a toxic psychosis that treatment in a general or mental hospital is the wisest course. Delays are dangerous. Early confusional states rapidly deteriorating may be overlooked.

If removal is impossible a naso oesophageal tube must be passed and a minimum of five pints of fluids given in the day. Vitamin B complex is best given by injection in the form of 5 mgm. aneurine hydrochloride and 50 mgm. nicotinamide three or four times during the day.

CONFUSIONAL AND DELIRIOUS STATES

As already emphasised these begin insidiously. Medical aid is often called in late as the early changes have not seemed remarkable. Physical causes should always be suspected. Pneumonia and pulmonary collapse are perhaps the commonest alcohol the most widely known. Besides treatment of the underlying condition which may not reveal itself at once adequate sedation and vitamin B therapy is called for. Paraldehyde m 180 repeated six hourly should be combined with 5 mgm. aneurine and 50 mgm. nicotinamide either by mouth or injection. Fluids must be pushed —up to five pints a day. For this reason alone these cases can be nursed at home only if (a) they are unfit to be moved or (b) a skilled nurse is available. A darkened room is to be preferred. Attention from several different persons is forbidden and rigid routine is essential.

If the case is taken early and the underlying cause is recoverable these cases do well. If the doctor feels that he cannot face the anxiety which these patients always cause early removal is a wise plan.

The possibility that these conditions may be caused by bromide intoxication should be borne in mind particularly in arterio sclerotic and elderly subjects. Two or three capsules each containing 20 gr. of sodium chloride should be given four hourly with large amounts of fluid in order to replace the toxic halogen by an inert one and encourage excretion of the bromide.

SUDDEN DELUSIONS AND HALLUCINATIONS

Sudden dramatic voicing of delusions or hallucinations may cause a family upheaval and a medical emergency. Careful

history usually proves that the patient has been slightly abnormal for a longer time or is passing into a confusional state such as we have just described. The treatment in the first instance should be as for that condition with emphasis upon the likelihood of mental and physical exhaustion. It is as well to warn the relatives that removal may be called for in a very short time.

ACUTE HYSTERICAL DISORDERS

There is no clear dividing line in the writer's opinion between psychotic and psychoneurotic states; they merge into one another. But we have still to consider conditions which are classified as hysterical: sudden paraplegias, collapses, fits and attacks of extreme pain. Great alarm is caused and one is left with the impression that the patient is insisting on creating consternation and dismay. Both he and his relatives require relief usually from each other and not always by sedation. The doctor has the difficult task of prevailing upon an already excited household that fuss is not called for and that the patient must quietly submit to the doctor's plans. These will entail temporary segregation of the patient from others and his being kept in bed. When family quiet is restored a hypnotic is called for such as two capsules of sodium amytal gr 3 given with a large drink. The patient is then left for four hours. Paresis will not be cured by this method but dramatic attacks will not recur at once. When there is more time the cause of the disturbance must be investigated upon lines suggested in psychiatric text books.

Mild or gross hysterical phenomena are compromise solutions of two or more alternative plans of action *e.g.* the wish to succeed in an over-ambitious course and the realisation that success is impossible. In such instances the symptom is both the excuse and the escape. In some instances the patient could not tolerate the sudden conscious recognition of the problem *e.g.* where a fugue has prevented a suicidal impulse from being realised or recovery from monoplegia leaves the patient with no excuse for relinquishing unwelcome work. The sedation and quiet which we have recommended may result in the partial clearing of somatic symptoms and their substitution by fear or even panic attacks. If this occurs further heavy sedation is imperative or an immediate discussion of the case with a specialist who

MENTAL EMERGENCIES DURING PREGNANCY AND THE PUERPERIUM

(a) *EARLY* —The belief that a pregnancy cannot possibly continue is fairly common when the child is unwanted and the resulting conflict produces many symptoms of anxiety distress and hysterical complaints. But as is well known these phenomena do not justify termination of the pregnancy. Such mothers are usually clamant in their demands revealing a past history and often a family history of poor social adaptation to stress and strain changed jobs frequent scenes and hysteroid behaviour. A very different picture is presented by a woman with a history of previous psychosis who becomes deeply depressed voices delusions or presents hypomanic behaviour. The same is true of these symptoms when they occur with no previous history of similar disorder. If there is the least doubt early admission into a general hospital or psychiatric clinic is desirable so that a full survey of the position may be made.

It is important to realise that persons of the first type the hysteroid usually accept the situation when the medical position is made clear and when relatives co operate. Suicidal gestures may occur but if the relatives will co operate the danger is much lessened. Usually when the husband understands that termination cannot be contemplated the patient herself becomes composed. The difficulties in differentiating the second type are considerable and specialist advice should be sought in doubtful cases. If a tragedy occurs it is more lamentable because the true psychotic conditions have a good recovery rate. As a general rule termination is to be advised where there is a bad family or personal history of psychotic breakdown. The reverse is true of the socially inadequate with hysterical symptoms despite the eugenic paradox.

(b) *LATE* —Psychiatric symptoms in the later months of pregnancy are less common. They should always rank as potential emergencies and nursing away from home not necessarily in a mental hospital should be considered. Superstitious ideas that the child will be physically abnormal or mentally deficient are common enough. So also are mild obsessional rituals and fear excessive cleanliness fears of fire and fear of injuring the older children. More bizarre ideas a definite change

of mood to one of unwarranted elation, or retarded gloom should cause anxiety and call for adequate supervision

(c) **PUERPERAL**—Puerperal disorders, it is now realised, carry a far better prognosis than used to be believed, except in cases where the previous personality traits have been morbid. Adequate nursing is essential, a high fluid intake is necessary, and much greater care as regards limiting the number of visitors. Adequate help with the young baby is required. Omission of this precaution may arouse reactive psychotic disorders. So much do we now regard birth as a simple physiological process that we forget the mental re-adjustment that it calls for in a woman. An understanding, supporting environment should be hers. If it is absent, symptoms may show themselves in the psychiatric sphere.

The puerperal emergencies require the treatment we have described for the general types of reaction described above. The writer feels that breast feeding is often discontinued with too little reason though it has to be stopped in real emergencies. An uninterested mother should be persevered with; the infant must be protected from the hypomanic or agitated woman.

SUICIDAL RISK

'IS THIS PATIENT A POTENTIAL SUICIDE OR NOT? MAY HE BECOME SO?'—These questions are sometimes difficult to answer, and are best approached by a summary of those conditions in which suicide occurs.

(1) The depressions

A distinction is now made between **endogenous depression** (a melancholic illness arising usually without any precipitating factor and often occurring in a person whose family history gives evidence of similar attacks or manic episodes associated with loss of weight, loss of sleep, particularly after four a.m. a tendency for the patient to improve after midday with absolute regularity and little day to day variation otherwise) and **reactive depression** (grief, anxiety, and misery following an emotional or financial upset in a subject whose family and personal history is often clear, there is an associated loss of weight, difficulty in getting off to sleep at night, and often characteristic day to day variation with little or no difference between the early morning and evening moods).

In the endogenous disorders self blame is prominent in the reactive depression blaming others or circumstances is more usual. In the former dejection is marked in the latter tearfulness. Bereavement or financial loss may cause a severe reactive depression in individuals of good personality. In poorer types mild disappointment may arouse similar symptoms but they are apt to be more clamant and display much self pity. In both types suicidal gestures may occur and be successful. In the endogenous disorder the whole personality is deeply involved and unapproachable in the reactive discussion of the precipitating factors is usually practicable.

(2) Psychopathic personalities

The inadequate psychopath may commit suicide or threaten to do so. He does so for reasons that would deter the more stable. The poorer type of personality suffering from reactive depression merges with the psychopath. Some of these feckless persons get themselves into difficult circumstances and threaten suicide or insist on their relatives or others taking action (*e.g.* termination of an unwanted pregnancy refusal to live in a new house etc.) It is the occurrence of such cases—persons who cannot be regarded as continuously insane—which explains why the percentage of the true psychotics amongst suicides is lower than we expect.

(3) The deluded and hallucinated

Here suicide is the logical outcome of illogical ideas or of aural hallucinations telling the patient to act in a way that must imperil life. Ophelia's death was probably so determined.

(4) The confused and delirious

If they are physically strong enough these patients may be so disorientated or subject to false conclusions that they swallow poison in mistake for simple drinks or go through windows in mistake for doors.

There are other patients frequently deemed to be suicidal namely those who are *actually* suffering from obsessional anxiety and in whose minds the *idea* that they might commit suicide (as indeed we all *might* do) is regarded as tantamount to the danger that they will. A sufficient discussion of these matters will clear up the doubt in our mind. Obsessional anxiety

may lead on to an acutely agitated state which must be dealt with appropriately. It is very desirable to distinguish these types but it may call for a specialist's opinion.

How then are we to assess the risk and treat the patient? A family history and personal history are most important and enable the doctor to show the family his doubts as well as his determination to take adequate precautions. A family history of suicide should make one doubly cautious as it undoubtedly increases the risk. Whilst the previous standards of an individual give some indication the most rigidly religious person in severe depression may intensely mourn — That the Everlasting had not fixed His canon 'gainst self slaughter — and find it impossible to resist the temptation to end it all. The nearer to the psychopathic inadequate type we come the more likely is a frightening gesture to occur particularly if the individual believes that he can gain his ends thereby.

Nothing is to be lost by asking the patient whether he has considered suicide or not. It is some relief to the deeply depressed and it reveals the social outlook of the psychopathic type. It enables one to gain a clear insight into the patient's difficulties and to modify his ideas.

In all doubtful cases insist on transfer to hospital. This is particularly important where self blame is marked and where there is severe constitutional upset or such withdrawal from reality as denotes a stupor or confusional state. In those cases where the patient is kept at home adequate sleep is essential and too much sleep is better than too little. Slowly acting barbiturates such as sodium barbitone (medinal) gr 10 on retiring should produce six hours rest.

The greatest difficulty exists in cases of reactive depression particularly when self pity and theatrical behaviour are suspected. When the symptoms are overdrawn a short period in an environment distasteful to the patient such as a mental observation ward is no bad thing and necessary arrangements can be made. Where the patient threatens suicide for purposes of getting his own way the practitioner must stress the point of view that the choice is the patient's own and to the relatives that giving way will not ultimately help the person and that the risk may have to be run. Such an attitude is usually successful if the relatives co-operate. When they do not their submission will temporarily

please the patient but the ultimate results will be less good. There is no harm in offering mild sedatives in this case, but quiet emphasis that the patient's attitude is really one of black mail is desirable. In these cases we are dealing with those who are acting not as adults but with the egotism of spoilt children. As in cases of hysterical outbursts with them an opportunity must be taken to get the patient to express his point of view, and then for sympathy to be expressed as far as possible combined with firm handling.

LEGAL ASPECTS OF PSYCHIATRIC EMERGENCIES

The practitioner faced with an acute mental emergency may ask himself, and be asked, a number of medico legal questions.

The position of the medical practitioner has been helped by the last extensive legal enactment about persons mentally disordered, *i.e.*, The Mental Treatment Act, 1930. Provided that the doctor can show that he acted in good faith no action at law will be successful against him. In future therefore the interpretation of this Act will probably be a wide one, and the medical man's anxieties now centre more upon the question— Should this patient be removed from his home or not?

He should be cared for elsewhere if sufficient nursing care and supervision cannot be provided. If he commits suicide seriously injures others or dies of malnutrition the medical practitioner may well be held to blame. The possibility of these disasters is more real than many practitioners realise. Also they are often much more imminent than relatives admit for they often feel that they themselves can only keep calm with a stiff upper lip and this they think implies minimising danger. If the decision is made to keep the patient at home provide adequate nurses. Male nurses may be required and a suitable Nurses' Co-operation should be asked to supply them. Except in the case of extremely excited or impulsive men trained women are as successful with men as are male nurses.

If the relatives will not accept the doctor's advice to remove the patient or have adequate attention, it is wise to ask them to sign a note saying that they are taking this responsibility. This need not lead to unpleasantness it underlines the doctor's advice which may well be acted upon shortly. The danger of inadequate feeding at home is quite as common as the danger

of suicide or extreme violence and its effects may mean the difference between short illness and chronic disablement
If the patient is to be removed, how can this be done?

Any person can be removed by the Relieving Officer who will also provide an ambulance and nurses after he is satisfied with the doctor's verbal assurance or written word— Mr X Y of is suffering from a mental disturbance sufficiently grave for him to need care at once. If you must call him in ring up the police who will inform you of his telephone number. In all questions of doubt the local superintendent of police can be relied upon to help.

Although any person may be thus removed and detained in a mental observation ward on a three day order signed by the Relieving Officer there are many cases where this is repellent to both doctor and relatives. General hospitals with psychiatric units may be prepared to admit urgent cases. Failing this a nursing home used to dealing with such cases or a special hospital must be sought. Good nursing homes are not numerous and indifferent ones are to be eschewed however clamorous the relatives are against a mental hospital. All satisfactory nursing homes which cope with the mentally disordered are more expensive than mental hospitals. The writer believes that although the relatives or patient may prefer the nursing home reasonable pressure can be properly brought on the relatives to choose the hospital except when money is of little moment. Much financial anxiety is caused to relatives who have chosen the nursing home and find the patient faced with a longer illness than they expected.

Direct admission to a mental hospital without preliminary reception into the mental observation ward must be arranged specially. If the patient's means are limited and he will be a rate-aided patient it must be clear that he belongs to the district served by the hospital selected. The same is true for patients of the private class entering a large borough or county hospital. This problem does not arise where the smaller hospitals for private patients are concerned. These make charges of four guineas a week upwards (except the Royal Asylums in Scotland which benefit from large endowments).

In cases where the Relieving Officer and mental observation ward are eschewed four methods of admission are possible and

the choice made before the hospital authorities are telephoned. The patient may be admitted —

(1) AS A VOLUNTARY PATIENT — He signs a form applying for admission on reaching the hospital

(2) ON AN URGENCY ORDER — This is an authorisation by a near relative asking for the patient's admission and accompanied by one doctor's certificate

(3) AS A TEMPORARY PATIENT — This is only applicable to patients who are unable to express themselves as willing or unwilling for treatment but is very suitable for the stuporose, confused and delirious cases. Form A 1 (Mental Treatment Act 1930)* is completed, consisting of a request for admission from a relative and two statements by doctors who must not be partners, one of whom must be specially approved to advise re temporary patients. All specialists in mental disorders are so qualified and have the necessary form available.

(4) AS A CERTIFIED PATIENT — This is a method too slow for emergencies and will not be discussed further.

Only in a few emergencies will voluntary admission be possible because of the patient's unco-operative behaviour. Since he may refuse to sign the approved form for voluntary admission when he arrives, it is often wise to give the relatives an Urgency Order to be used in case of necessity on reaching the hospital so as to avoid any further delay. Urgency Orders and Temporary Orders may be signed by responsible persons if no relatives are available (e.g. a nursing home matron, hotel manager or close friend).

It will be noted that neither 1, 2, or 3 involves certification. This is a great load off the mind of a layman who regards certification as implying a life-long stigma. This connotation is deplorable for certification really implies that the patient is so unwell that he cannot co-operate and someone else must make decisions for him. Emphasis on this point to the relatives is of great avail in modifying their repugnance if certification becomes necessary later. In the acute stage with which we are dealing, certification does not arise but may well

* Although psychiatric emergencies are not common, 1 or 2 Urgency Orders (called Lunacy 4 & 2) may well be carried in the doctor's car. These (and Form A 1) are obtainable from Messrs. Shaw & Sons, 7 Fetter Lane, London, E.C. 4 (Tele. CENTRAL 8171).

do so after the patient arrives at the hospital if as a voluntary patient, he is obviously very ill and insists on leaving, or if, when received on an Urgency Order, he is too ill to leave within seven days. An Urgency Order is only valid for this period.

One is then in a position to telephone the proposed hospital and arrange how the patient is to be moved and how supervised during the removal (male or female nurses). The hospital should be given details about the sedatives that have been given before removal. If the hospital and the relatives understand the matters clearly, and particularly what papers are necessary, the doctor need not remain in the house any longer.

HENRY WILSON

CHAPTER XIII

Medical Emergencies in Diabetes

KETOSIS

HYPERGLYCÆMIA is the cause of many of the symptoms in diabetes but never alone constitutes a medical emergency the presence of excess of ketone bodies in the blood however must always be looked upon as a potential if not actual source of danger Ketosis is due to the accumulation of beta hydroxy butyric acid and diacetic acid in the blood in amounts sufficient to allow of their excretion and recognition in the urine Beta hydroxybutyric acid forms about 70 per cent of the ketone bodies in the urine but its presence there cannot be recognised by either Rothera's (ammonium sulphate sodium nitroprusside and ammonia) or Gerhardt's (ferric chloride) tests The remaining 30 per cent is made up almost entirely of diacetic acid acetone being present in the urine only in traces From a practical point of view diacetic acid is by far the most important and can be detected in the urine by Rothera's test in dilutions up to 1 in 400 000 acetone also gives a positive reaction in dilutions up to 1 in 200 000 Gerhardt's ferric chloride test gives a positive reaction with diacetic acid only up to the much higher concentration of 1 in 2 000 and is negative to acetone It follows therefore that when testing for ketone bodies in the urine Rothera's test should be done first and if this is strongly positive the ferric chloride test should also be carried out The presence of a positive ferric chloride test must be regarded as indicating severe ketosis and the possibility of the patient's passing into precoma or coma unless promptly and suitably treated

Symptoms

Severe degrees of ketosis may occur without any symptoms or signs referable to this condition but more often the patient complains of one or more of the following shortness of breath anorexia nausea vomiting drowsiness and abdominal pain Vomiting is particularly important as it tends to set up a vicious circle in which starvation increases the ketosis and in turn

aggravates the vomiting with a result that the state of severe ketosis may swiftly progress to diabetic coma. Abdominal pain especially when associated with vomiting may present a very real diagnostic problem the importance of which is accentuated by the fact that the unnecessary opening of the abdomen of a patient in severe ketosis may greatly prejudice his chance of recovery. The abdominal pain of ketosis is usually generalised and diffuse there is little or no rigidity and signs of peritoneal irritation are absent (*See also page 38*). Rarely there may be marked upper abdominal distension due to acute dilatation of the stomach which may complicate the picture and necessitate appropriate treatment by lavage and continuous suction drainage.

Causes

The danger of continued vomiting in severe diabetes has been pointed out and it follows that any condition which produces it must be regarded as a potential cause of diabetic coma. The danger of starvation in this connection has also been explained and if absence or deficiency of insulin be added the conditions most favourable for the production of coma are complete. It is not surprising therefore that diseases affecting the gastro-intestinal tract particularly gastro enteritis stand high on the list of causes of diabetic coma not only because they are so often associated with vomiting and starvation but also because it is common for diabetics suffering from them quite erroneously to conclude that inability to take food is a contra indication to their giving themselves insulin. Other causes include infections outside the alimentary tract such as septicaemia pyelitis and pneumonia local infections such as carbuncles infected gangrene and insulin abscess and the stopping of insulin.

It is important to realise that although ketosis may clear up spontaneously it is more likely unless treated to increase in severity until it gives rise to coma and ultimately death. It follows that in the treatment of diabetes every effort should be made to prevent ketosis and when it occurs to treat it before the ferric chloride test becomes positive. If every diabetic with severe ketosis were regarded as a potential case of diabetic coma and appropriately treated before the onset of symptoms the incidence of this most dangerous but usually most preventable medical emergency could be greatly reduced.

Treatment

The treatment of severe ketosis will therefore be considered under the heading of the prophylactic treatment of diabetic coma

DIABETIC COMA

There is no hard and fast dividing line between severe ketosis and pre coma and the latter term is best reserved for those cases which present the clinical manifestations of ketosis already described but although drowsy retain consciousness. The term diabetic coma is often loosely applied to this latter group of cases but should be reserved for those in whom there is actual loss of consciousness

Diagnosis

The first step in making a diagnosis of diabetic coma is to obtain from relatives or friends as detailed an account as possible of events immediately preceding the onset of coma. If such an account is available more often than not the diagnosis will be apparent before the patient is examined. This is no less true of patients in whom the disease is first diagnosed in coma than in those known to have diabetes prior to the onset of coma. In the former a history of thirst polyuria and loss of weight preceding the onset of coma will focus attention in the right place when it comes to examination. If the patient is known to be a diabetic on insulin the diagnosis is most likely to be diabetic or hypoglycæmic coma the value of history in distinguishing between these two conditions cannot be over emphasised and will be referred to again later

The onset of diabetic coma is gradual and follows a period of severe ketosis sudden loss of consciousness without the premonitory symptoms and signs of pre coma is rarely if ever due to diabetic coma. The signs are a combination of those produced by ketosis and dehydration the former include furred tongue acetopnœa and deep abdominal respiration and the latter lowered ocular tension dry tongue and skin rapid pulse and low or falling blood pressure. Rarely there may be a state of restlessness which may be so intense as to simulate actual mania

Examination of the urine shows heavy glycosuria and intense ketosis the ferric chloride test being strongly positive. The urinary chlorides are typically diminished or absent. Very occasionally there may be complete absence of sugar and ketone

bodies in the urine associated with severe impairment of renal function and the passage of large numbers of casts in such cases the secretion of urine may fall or even cease and the blood urea reach very high figures. The blood sugar is invariably raised and usually exceeds 400 mgm per cent although diabetic coma may rarely occur with lower levels of 300 upwards. The ketone bodies in the blood are also greatly increased and although not usually estimated may be detected by applying Rothera's test to a few drops of plasma or serum in a watch glass a useful procedure in the rare cases referred to above in which renal damage results in absence of ketonuria.

If hypoglycæmia is excluded the commonest differential diagnosis of diabetic coma is a lesion in the central nervous system such as hæmorrhage or thrombosis occurring in a diabetic. In such cases, starvation and the withholding of insulin may result in glycosuria and ketosis but the latter is usually much less severe than in diabetic coma the patient being typically elderly and therefore often only mildly diabetic. Localising signs in the nervous system such as hemiplegia are usually present subarachnoid hæmorrhage being an exception in this respect here again however ketosis is rarely severe and a lumbar puncture will establish the diagnosis.

Treatment of diabetic coma.

PROPHYLACTIC—For reasons already stated the treatment of severe ketosis and pre-coma will be described under this heading. Although the general principles of treatment will apply equally to both severe ketosis and pre-coma in practice the treatment of the latter is often rendered more difficult by the presence of vomiting and dehydration.

When ketonuria is the only clinical evidence of ketosis treatment depends to some extent upon whether the patient is already on insulin and if so on the type of insulin used and the arrangement of dosage. As a general rule any diabetic who shows severe ketonuria should be given insulin and if the ferric chloride test is positive it is wise to start with morning and evening injections of soluble insulin. The size of the dose will depend on the age of the patient and the severity of the disease as judged by its duration, the height of the blood sugar and the response

to diet if this has been tried in adults however it is usually a waste of valuable time to start with less than 20 to 30 units a day of which rather more than half should be given before breakfast. In untreated cases the effect of an adequate but restricted diet containing between 150 and 200 grammes of carbohydrate should be taken into account when assessing the initial dose of insulin.

In cases on diet and two injections of soluble insulin it may be sufficient to increase both doses but if this fails to produce a rapid reduction of ketonuria a third injection of 10 to 20 units according to the size of the morning dose should be given before the midday meal until the diabetes is again controlled. If the diabetes was previously controlled by one injection of protamine zinc insulin it is worth while trying first the effect of adding to it some soluble insulin and testing a specimen of urine passed at about six p.m. if this shows heavy glycosuria a small dose of 10 to 20 units of soluble insulin should be given before the evening meal.

Failure to clear the urine of ketone bodies in two or three days by this method or increase in the degree of ketosis indicates the necessity of stopping the protamine zinc or other delayed action insulin and giving two or three injections of soluble insulin in the manner described. Diabetics on one mixed dose of soluble and protamine zinc insulin should be treated in the same way first by the addition of soluble insulin before the evening meal and if this fails by the use of three or more injections of soluble insulin.

In this group of diabetics provided that the diet contains an adequate carbohydrate content of not less than 150 and preferably 180 to 200 gm per day no change need be made although it is advisable to make sure that the intake of fat does not exceed 100 gm per day.

In pre coma a state of actual emergency exists and it is always wise to discontinue delayed action insulins if in use and rely entirely on multiple injections of soluble insulin. For the treatment of this class of case it is helpful to have some standard scheme which can be readily understood by the nurse or other person in charge and which can be instituted with the minimum of delay such a scheme has been worked out by Lawrence and is called by him— The Emergency Sheet (Fig 17)

Soluble insulin should be given four hourly at times recorded on the sheet with carbohydrate two hourly in one or more of the suitable forms listed in the top right hand corner. The dose of insulin will depend on the colour obtained by testing a four hourly specimen of urine with Benedict's solution and should be prescribed in the space provided. It is impossible to be dogmatic about doses of insulin but in a diabetic of moderate severity and average insulin sensitivity it may be of the order of 28 to 40 units for a red or yellow test 12 to 20 for a thick green and 0 to 8 for a blue the use of a small dose of insulin in severe diabetics even when the urine is sugar free often prevents a serious relapse by the time the next test is made.

If 20 gm of carbohydrate are given every two hours the patient will receive 240 gm in 24 hours which should be sufficient to cover the insulin and replenish the liver glycogen later 40 gm may be given at four hourly intervals and the insulin dosage modified to allow for the increasing insulin sensitivity associated with diminishing ketosis.

Dehydration may be a marked feature in pre coma especially if this condition has been brought about by continued vomiting and should be treated *simultaneously with the ketosis*. In the absence of repeated vomiting the less severe degrees of dehydration may be corrected by two hourly fluid feeds alone or with the addition of water by mouth between feeds but before adopting this method of treatment it is a good plan to wash out the stomach with a weak solution of sodium bicarbonate (one teaspoonful to a pint). Severe dehydration with a low or falling blood pressure and decreased ocular tension to mention only two of the more important signs should be treated by immediate intravenous saline infusion as in established coma the rule being when in doubt give fluids intravenously. Much valuable time may be lost and recovery delayed if not jeopardised by trying to give fluids orally to severely dehydrated precomatose diabetics such efforts are liable not only to prove ineffectual but also to produce vomiting and so aggravate both the dehydration and the ketosis.

No less important than the treatment of ketosis and dehydration is that of the underlying cause of the condition. Mention of this has been left until last because in most cases such treatment must necessarily follow the institution of the measures already

described while in the comparatively few instances in which this is not so as for example when the cause is an insulin abscess treatment is surgical and will be considered under the heading of operations. Where ketosis is associated with infection by an organism susceptible to some specific form of therapy such as penicillin the appropriate treatment should be instituted as soon as possible.

Removal to hospital

THE TREATMENT OF ESTABLISHED COMA WITH BLOOD SUGAR CONTROL—Every case of diabetic coma needs constant skilled attention and should therefore be treated in a hospital or nursing home. Should a diabetic go into coma at home a difficult decision may have to be made as the more severe and prolonged the coma the greater the need for hospital treatment the greater also is the risk of moving the patient. It is impossible to lay down hard and fast rules for every case but certain considerations are of special importance in reaching a decision which may mean life or death to the patient. Obviously the home conditions availability of doctor and nurses and proximity of a suitable hospital have a direct bearing on the problem but perhaps the most decisive single factor is the patient's blood pressure. If the systolic pressure exceeds 100 it is usually best to move the patient immediately to hospital if the figure is much below this level the possibility of improving the patient's condition before moving him must be seriously considered. The best way of doing this is to give a moderate dose of 40 to 60 units of insulin and a rapid intravenous infusion of two litres of normal saline after which the patient will often be able to stand the journey to hospital without the fatal fall in blood pressure which is liable to take place if this preparatory treatment at home is not carried out.

Warmth

On admission to hospital the patient should be put into a warmed bed and kept warm with an electric blanket all other methods are dangerous and less effective hot water bottles being particularly liable to produce burns unless suitably protected. The foot of the bed should be raised and blood pressure recorded the cuff being left in position for subsequent readings at hourly intervals.

Urine and blood sugar.

A catheter specimen of urine should next be taken and examined for sugar ketone bodies and chlorides and a sample of blood for sugar estimation

Insulin

While all this is being done 80 units of insulin should be injected half being given intravenously if there is marked circulatory collapse and an intravenous drip set up if possible using an arm vein without cutting down. The reason for this is that infusion of glucose solution in the writer's experience often causes *phlebitis* when given into an ankle vein especially if a cannula is tied in

Saline

Opinions differ as to the best solution for intravenous infusion in the early stages of diabetic coma, but normal saline is probably the most satisfactory as it corrects dehydration hæmo concentration and chloride loss without the disadvantage attached to the use of glucose solution of masking the effect of the initial dose of insulin. If chlorides are completely absent from the urine (*see page 304*) 2 per cent or even 5 per cent saline can be used with advantage until they re-appear. The first litre should be allowed to run in by gravity as fast as possible and if dehydration is very severe a second litre can safely be given in the same way after which the rate of flow should be cut down to a drip

This should be continued until the patient is well able to take fluids by mouth without vomiting in this connection it is important to remember to wash out the stomach early in the treatment of diabetic coma (*see page 391*)

Dextrose

As soon as the blood sugar begins to show a definite fall it is wise to change over to 4 per cent dextrose in one fifth normal saline and to continue with this solution until the drip is taken down. If the blood pressure continues to fall the substitution of plasma for saline will sometimes prevent death from peripheral circulatory failure and is always worth a trial. Extract of supra renal cortex has been advocated in this connection but although the writer has used it on a number of occasions the results have never been impressive

By the time the drip is set up the initial blood sugar should be known and the insulin dosage planned accordingly. If the figure exceeds 1 000 mgm per cent a further 100 units may be given two hours after the first injection if it is between 750 and 1 000 mgm per cent 50 units but if less than 750 mgm per cent it is probably better to withhold insulin until the result of a second blood sugar is available. This should be taken three hours after the first so that the level may be known in time to give insulin four hours after the initial injection. The dose of insulin then given will depend on the response to treatment in the first three hours and the importance of knowing this constitutes the chief reason for giving saline rather than glucose intravenously over this critical period. The tendency for the intravenous fluid to lower the blood sugar by dilution should be allowed for in assessing the response of the blood sugar to the insulin already given. If at the end of three hours the blood sugar equals or exceeds the original figure but is less than 1 000 mgm per cent a further 100 units should be given 40 units intravenously 60 units subcutaneously this dose should be doubled if the blood sugar exceeds 1 000 mgm per cent. For a poor reduction of 100 mgm per cent or less 80 units should be injected and for a satisfactory fall of over 100 mgm per cent 50 units or less according to the blood sugar. Subsequent dosage can be worked out in the same way from regular blood sugar estimations taken in conjunction with the results of four hourly tests for sugar and ketone bodies in the urine a catheter being allowed to remain in the bladder for this purpose. As soon as ketosis has been significantly reduced the patient's sensitivity to the insulin is likely to increase and the dose of insulin must be correspondingly reduced so as to avoid hypoglycæmia. The later stages of treatment are identical with those described under pre coma the Emergency Sheet being used in both cases.

TREATMENT OF ESTABLISHED COMA WITHOUT BLOOD SUGAR CONTROL—No mention has been made of the treatment of diabetic coma without blood sugar control as this should be necessary only in exceptional circumstances. The general principle is the same but the dosage of insulin must be decided by the results of three or four hourly urine tests taken in conjunction with the patient's clinical condition. In the early stages ketonuria is more helpful

than glycosuria which will be persistently heavy the quantitative value of the ferric chloride and nitro prusside tests described at the beginning of this chapter being of great importance in this connection. The chief difficulty lies in deciding the size of the first few doses of insulin and one initial blood sugar is of great importance and assistance in this respect.

As in pre coma the draining of local collections of pus if it involves no major surgery should be carried out as soon as possible but major operations should be postponed until the patient is out of coma.

A state of drowsiness may persist for many hours after ketone bodies have disappeared from the urine and tachycardia may also be present for several days the latter is probably due to myocardial intoxication and should be treated by complete rest. It has been suggested that the cardiac condition may be the result of vitamin B₁ deficiency and daily injections of 25 mgm have been advocated for its treatment.

Sudden cardiac failure may occur in the diabetic recovering from coma particularly if roughly handled. Gentleness in nursing is very important and it is wise not to sit the patient up until the pulse rate returns to normal.

HYPOGLYCÆMIA

The blood sugar level at which symptoms and signs of hypoglycæmia make their appearance varies in different diabetics but is typically not higher than 60 mgm per cent unless possibly the rate of fall is very rapid. There is a time relationship between the onset of hypoglycæmia and type and dosage of insulin injected the larger the dose the longer the period during which hypoglycæmia is liable to occur. With soluble insulin in small or moderate doses of up to 30 units the maximum fall in blood sugar may be expected from three to six hours after injection but with doses of over 40 units hypoglycæmia may be delayed for as long as 10 or 12 hours. With protamine zinc insulin the time interval is longer hypoglycæmia after a morning injection occurring sometimes in the late afternoon but more often during the night or early morning. Globin insulin resembles protamine zinc insulin but its action is less prolonged with the result that moderate doses tend to produce their maximum fall in blood sugar about 8 to 12 hours after the injection. In some

cases however hypoglycæmia may occur before the midday meal.

Symptoms

The onset of hypoglycæmia is always rapid a matter of minutes or at the most an hour or so the patient being typically in his usual state of health immediately prior to the attack this fact alone is sufficient to differentiate between hypoglycæmic and diabetic coma in almost every case where an accurate history can be obtained

The early symptoms are due to the secretion of adrenaline and include palpitation sweating tremor restlessness and excitability The low blood sugar is commonly associated with intense hunger Hypoglycæmia by its action on the central nervous system may produce mental confusion incoordination ataxia paræsthesiæ especially in the lips and tongue transient palsies drowsiness coma convulsions and rarely death To this list may be added nausea and headache which are most commonly produced by protamine zinc insulin Coma is usually preceded by one or more of the early symptoms which although many and varied fortunately tend to be stereotyped in individual diabetics This enables coma to be prevented by treatment at the onset of the attack in all but the comparatively rare cases in which loss of consciousness occurs almost or completely without warning In such patients coma may occur without obvious evidence of its causation

Diagnosis

The importance of history of onset has already been stressed but if this is not available diagnosis must be made from the clinical picture and confirmed by response to treatment and if possible blood sugar estimation In hypoglycæmic coma the skin is pale and moist and the respiration similar to that of normal sleep the tongue is moist the blood pressure and ocular tension normal and heart rate increased Signs in the central nervous system may be entirely absent or there may be evidence of hemiplegia or other palsies with an extensor plantar response on one or both sides Epileptiform convulsions are not uncommon The urine contains no ketone bodies but sugar although often absent may be present if the bladder has not been emptied for some time previous to the attack On the negative side absence of signs of dehydration and severe ketosis

is particularly important in distinguishing between hypoglycæmic and diabetic coma. The main points in the differential diagnosis of these two states may therefore be summarised briefly as follows —

	<i>Hypoglycæmic coma</i>	<i>Diabetic coma</i>
Mode of onset	Sudden	Gradual
Respiration	As in sleep	Deep abdominal (Air hunger)
Acetopnœa	Absent	Present
Tongue	Moist	Dry and furred
Skin	Moist	Dry
Blood pressure	Normal	Low
Ocular tension	Normal	Low
Ketonuria	Absent	Heavy
Glycosuria	Variable	Heavy
Blood sugar	Less than 60 mgm per cent	More than 300 mgm per cent

Treatment

PROPHYLACTIC —Frequent attacks of hypoglycæmia mean bad diabetic treatment. They often result from an attempt to render the urine sugar free after meals instead of before them. The use of buffer feeds in the mid morning and at bed time will often prevent attacks and the latter should always be given to patients on protamine zinc insulin to reduce the risk of nocturnal hypoglycæmia.

THE TREATMENT OF THE ATTACK —This consists of giving the diabetic or more often in his taking glucose or some other rapidly absorbed form of carbohydrate such as sugar jam biscuit bread or chocolate in adequate amounts when symptoms begin. There is a general tendency on the part of diabetics and those treating them to give too little carbohydrate especially in attacks due to protamine zinc insulin with the result that either the attack is not relieved or recurs after a brief interval of improvement. Four lumps of sugar or their equivalent (20 gm of carbohydrate) is a reasonable initial dose and should be repeated in 15 minutes if symptoms persist.

With the onset of coma treatment at once becomes more urgent and less simple as carbohydrate food can no longer be swallowed. This difficulty can be overcome in a number of ways the best of which is to give concentrated dextrose solution intravenously if a 60 per cent solution is used 12 gm can be injected with a 20 c cm syringe which is sufficient to bring all but the most severe cases out of coma in a few minutes. If sterile glucose solution is not available and coma is not too deep 10 minims of 1 in 1 000 adrenaline subcutaneously may revive the patient sufficiently for sugar to be given by mouth. Posterior pituitary lobe extract has a similar action in raising the blood sugar but is slower and is unsuitable for pregnant diabetics. A solution of glucose or sugar may be given by means of a stomach tube or nasal catheter the former being the safer method for those not experienced in the art of nasal feeding. Rectal glucose is inefficient in raising the blood sugar and should not be relied upon in the treatment of hypoglycæmic coma. It is scarcely necessary to add that though sucrose is effective by mouth it is useless to give it intravenously or by rectum.

When consciousness has been regained the patient should be given a carbohydrate feed and carefully watched for evidence of relapse.

A small number of cases of profound and prolonged coma fail to respond to elevation of the blood sugar and either die or only slowly regain consciousness after a period of many hours or even days such cases are liable to exhibit temporary or permanent mental and nervous changes from damage to the brain.

In conclusion whenever there is the least doubt about the cause of coma in a diabetic it is a safe rule always first to give glucose without insulin and if coma is due to hypoglycæmia rapid recovery is likely to result if it is diabetic no harm will be done.

OPERATIONS

Minor operations

If only a local or brief general anæsthetic such as gas and oxygen or thiopentone (pentothal) is required operation can be carried out with little disturbance to the diabetic regime. The usual insulin and diet should be taken up to the last meal before the anæsthetic and this should consist only of the carbohydrate portion. Sufficient time should be allowed after this meal for

the stomach to empty and an extra 15 gm of glucose given as a small drink about one and a half hours before the operation

Major operations

While almost any anæsthetic except chloroform may now be given to a diabetic providing that the diabetes is reasonably well controlled and the patient suitably prepared the shorter the period of unconsciousness following the operation the smaller will be the risk of post operative ketosis. Anæsthetics such as ether however which tend to cause post operative vomiting are best avoided. When severe ketosis is already present unless immediate operation is necessary to save life it is wise to treat the patient according to the Emergency Sheet until the ketosis has been cleared up before an anæsthetic is given by so doing the risk of diabetic coma following the operation is greatly diminished. When operation is not a matter of urgency the patient should first be stabilised on two or more injections of soluble insulin and kept on it until the usual diet is resumed.

On the day of operation the procedure will depend on the time the operation is to take place. The possible alternatives are too numerous to consider in detail but the general principle is to give enough carbohydrate and insulin before operation to build up an adequate supply of liver glycogen and at the same time send the patient to the theatre with a relatively empty stomach and not too high nor too low a blood sugar. If the operation is timed for 10 a.m. the usual morning dose of soluble insulin may be given at 8 a.m. together with about 20 gm more than the usual breakfast carbohydrate in the form of a glucose drink. When the time is noon the morning dose of insulin is given at 7 a.m. followed by the usual breakfast carbohydrate in the form of toast and marmalade and 30 gm of glucose in 4 fl oz of iced orange juice at 10.30 a.m. If the operation is at about 4 p.m. no change in the diabetic treatment need be made until the midday meal which should consist of the carbohydrate portion only and be given as a glucose drink at 2 p.m. together with an extra 10 to 15 units of insulin. These methods of preparation are to be considered as suitable only for patients with mild or moderately severe diabetes difficult and very severe cases requiring expert individual management.

In all major operations, however, sterile dextrose should be available for intravenous injection in case the patient vomits the glucose drink prior to, or during the induction of the anæsthetic, and a pint of 5 per cent glucose should be given routinely as a rectal drip as soon as the patient leaves the theatre

WILFRID OAKLEY

CHAPTER XIV

Medical Emergencies in Other Endocrine Disorders

THYROID

Toxic goitre.

THE emergencies which may arise in a patient with toxic goitre are the thyroid crisis and congestive heart failure which may or may not be associated with a crisis

(a) Thyroid crisis.

This condition may occur either during the course of the disease or after operation and is characterised by a rapid increase in the symptoms of thyrotoxicosis. The patient becomes restless and irritable, is sweating and flushed, and often delirious. The pulse rate may rise to 200 or more and there is a rise of blood pressure and temperature which may be as high as 106°. Pulmonary œdema may be present. Later the patient may become collapsed with a pale, cool clammy skin and death is the result in most cases, often within a few hours of the onset.

The mechanism of a crisis is obscure. It may be precipitated by psychological disturbances. It has been suggested that an excessive secretion of adrenaline is responsible since the symptoms are comparable with those of an overdose but there are probably other factors. Since the fever is not due to infection there is evidently a breakdown in the heat regulating mechanism. One hypothesis that has been suggested is that the condition is caused by acute thyroid failure but the syndrome certainly does not occur in myxœdema.

PREVENTION.—In the management of a severe case of toxic goitre all precautions should be taken to prevent a crisis, because once it occurs the outlook is grave. Rest in bed, the avoidance of psychological upsets, a high calorie diet with abundant carbohydrate and the administration of a sedative such as phenobarbitone gr $\frac{1}{2}$ three times a day, are important. Operation on such a patient should never be "rushed" but should only be

undertaken when the maximum improvement has been obtained with medical treatment. The incidence of crisis has fallen considerably since the introduction of the pre operative use of iodine. There are however cases of toxic goitre which do not improve with medical treatment but continue to deteriorate. Careful judgment is necessary to decide when to operate on such patients. In all severe cases in order to reduce the risk of crisis the operation should be performed in two stages and in those where response to medical treatment has not been satisfactory ligation of the thyroid arteries should be performed some time before lobectomy is attempted. The two stage operation is less necessary now since the advent of methyl thiouracil. Throughout treatment the winning of the confidence and co operation of the patient is essential.

Treatment—The importance of the correct management of toxic goitre has been stressed because it is so great a factor in preventing a crisis. Should this occur a large dose (60 minims) of Lugol's Solution (Aqueous Solution of Iodine B P) in milk should be given at once followed six hours later by 30 minims and repeated six hourly for the next 24 hours. If there is much restlessness and mental disturbance (and this is usually the case) 3 grains of phenobarbitone should be given by mouth or $\frac{1}{2}$ of a grain of morphine subcutaneously. The patient should be kept cool with ice packs and the early and continuous administration of oxygen is usually beneficial. Since full stores of glycogen in the liver are said to reduce the incidence of crises 10 per cent dextrose in 500 c cm of normal saline should be given intravenously and be repeated at intervals. Some authors recommend the addition of 5 to 10 grains of sodium iodide to the intravenous dextrose saline.

(b) Congestive heart failure

This occurs as a result of the added burden of thyrotoxicosis on an already damaged myocardium. Failure may or may not be associated with auricular fibrillation. Acute congestive failure in toxic goitre should be treated on the same lines as congestive failure unconnected with thyrotoxicosis but it should be remembered that the thyrotoxic patient has a greater sensitivity to drugs such as morphine which should therefore be given in slightly smaller dosage. If failure is immediately endangering the life

of the patient Lugol's solution should be given at once in order to diminish the degree of thyroid intoxication instead of postponing it until the full effect of digitalis has been obtained. During the first 24 hours 30 minims should be given six hourly and thereafter 10 minims three times a day.

(c) Acute thyrotoxic myopathy (Acute thyrotoxic bulbar palsy—Laurent)

This is a very rare condition which may occur in severe thyrotoxicosis. It is a rapidly spreading bulbar palsy with paralysis of the muscles of mastication and deglutition, facial weakness, ptosis, diplopia, paresis of the muscles of the neck and generalised weakness of the limbs.

It is practically always fatal within one or two weeks of onset. Prompt relief of the paralysis occurs after the subcutaneous injection of prostigmin and in this the condition resembles myasthenia gravis.

J. H. Sheldon and R. Milnes Walker* have described a case in which treatment with prostigmin and partial thyroidectomy was followed by rapid recovery. After the initial subcutaneous injection of 1 mgm of prostigmin the drug was given by mouth in doses of 15 mgm four times a day. After the partial thyroidectomy it was continued in doses of 90 mgm daily by mouth and gradually reduced.

Substernal goitre

Unless it is substernal simple goitre does not endanger life. Long standing nodular goitres may rapidly increase in size through sudden hæmorrhage into a cyst. If this occurs in a substernal goitre sudden pressure on the trachea may cause asphyxia, coma and death. On examination part of the enlarged gland may or may not be palpable above the sternum but there is always dulness on percussion over the manubrium. In less urgent cases an X-ray examination should be made and will reveal a substernal mass and deviation of the trachea. Treatment consists in immediate partial thyroidectomy.

PARATHYROIDS

Tetany is the name given to a state of increased neuromuscular excitability consequent upon a reduction in the

**Lancet* 1916 1 34

concentration of ionised calcium in the tissues. The condition may be associated with a variety of diseases but the clinical picture is essentially the same in them all—muscular twitchings (especially on percussion) carpo pedal spasm drowsiness stridor and in severe cases even convulsions and coma.

The underlying causes may be placed in two groups. Firstly those represented by rickets osteomalacia coeliac disease idiopathic steatorrhoea renal dysfunction and hypoparathyroidism either following operation or occurring spontaneously. In all of these there is a diminution in the total serum calcium and a proportionate fall in the amount of available or ionised calcium. Secondly there are cases in which the total calcium in the serum is normal but on account of an alkalotic state in the tissues too little of it has ionised and become available and thus patients suffering from persistent vomiting (loss of chloride) hyperventilation with excessive depletion of volatile acid (carbon dioxide) as in hysterical hyperpnoea and prolonged overdosage with easily diffusible alkali (e.g. bicarbonate of soda) fall into this group.

The emergency treatment of tetany when it is associated with low serum calcium is to raise the level of calcium by parenteral injection. In urgent cases i.e. those with painful spasm of the limbs and severe laryngeal spasm calcium gluconate 10 to 20 c cm of a 20 per cent solution should be slowly injected intravenously. In less urgent cases with mild spasm of the hands and feet only it may be given intramuscularly. Calcium chloride 20 to 40 c cm of a 5 per cent solution may also be given intravenously but it has the disadvantage that if any escapes into the subcutaneous tissues it may cause necrosis and ulceration. For this reason it should never be given intramuscularly. With the parenteral administration of calcium the spasms rapidly subside. Shortly after the intravenous injection it is as well to give an intramuscular injection of the same amount of calcium gluconate so that with its slower absorption the effect is prolonged. Thereafter the patient receives treatment for the chronic state and for the underlying cause. If the spasms recur the injections should be repeated. In very severe convulsions making intravenous injection difficult sedatives such as chloral hydrate gr. 30 by mouth or paraldehyde m 240 given rectally in ten times its volume of warm saline (110°F) may be necessary. Inhalations of chloroform might be used. These are only temporary measures to

quieten the patient and should not take the place of the administration of calcium

In parathyroid tetany which may arise 24 hours to one week after thyroidectomy the same treatment is adopted but in addition 20 to 30 units of parathyroid extract B.P. should be given intramuscularly 6 to 8 hourly. It acts by withdrawing the calcium from the bones thus raising the level of calcium in the blood but since it takes from 4 to 6 hours to achieve this it would not be effective in an emergency. In severe cases the first dose should be 50 to 60 units given intravenously. When parathyroid extract is used symptoms of hypercalcaemia—drowsiness increasing later to coma—may occur. Treatment consists in stopping the injections and reducing the increased viscosity of the blood caused by the excess of calcium by withdrawing a pint of blood and injecting one litre of normal saline intravenously. Parathyroid extract is not used in the treatment of chronic parathyroid tetany because it loses its effectiveness after a few weeks owing to the formation of an anti-hormone. At this stage however tetany can be kept in abeyance by giving calcium and vitamin D by mouth.

The essential treatment of tetany caused by alkalosis is to adopt appropriate measures to counteract the alkalosis and thereby increase the ionisation of calcium. Alkalosis caused by repeated vomiting is produced by excessive loss of hydrochloric acid and chloride in the vomitus and is treated by supplying chloride in the form of normal saline intravenously and quarter strength normal saline by mouth. When tetany arises through giving alkali the drug should be stopped and if this is not effective a few doses of an acid forming salt such as ammonium chloride 10 to 15 grains may be given but this is rarely necessary. In tetany caused by overbreathing the alkalosis is produced by the washing out of carbon dioxide from the tissues and the acute attack is treated by giving an inhalation of 7 per cent carbon dioxide in oxygen. If this is not obtainable the patient's expired CO_2 may be used by letting him re-breathe from a bag containing air or oxygen. The underlying hysteria should subsequently be treated.

ADRENALS

Crisis of Addison's disease

Patients with Addison's disease are liable to crises which are precipitated by exposure to cold undue exertion an intercurrent

infection or an operation. They may also be caused by an extension of the disease process. The early symptoms of a crisis are increasing weakness, vomiting, diarrhoea, mental changes and a fall in temperature and blood pressure. The serum sodium and chloride are usually low (normally 325 to 350 and 560 to 620 mgm per cent respectively) and the blood urea and non protein nitrogen practically always high (normally 20 to 40 mgm per cent). There is usually but not always hypoglycæmia. If untreated the patient develops profound exhaustion, dehydration and collapse and dies in coma. It is important to treat these cases early for if treatment is delayed the patient sinks into a refractory state which is unresponsive to therapy.

In a known case of Addison's disease the diagnosis of an *impending crisis presents no difficulty and should be suspected* if rapidly increasing weakness, vomiting or diarrhoea occur. In rare cases a crisis may be the presenting feature of Addison's disease. In these pigmentation of the skin may be absent or slight or the buccal patches may be overlooked and the condition may be mistaken for a severe gastro-intestinal infection. The finding of a low blood sodium is valuable in confirming the diagnosis and this estimation should be repeated at intervals in order to assess the results of treatment.

Treatment—The symptoms of a crisis are thought by some to be due to dehydration and by others to hypoglycæmia. In treatment therefore in addition to giving sodium chloride and cortical hormone it is important to combat these two conditions. As soon as possible a 5 per cent solution of glucose in 1.5 per cent saline should be administered intravenously by continuous drip. Two litres should be run in during the first 12 hours and 1 litre during the second 12 hours. While the continuous drip is being set up 50 c cm of suprarenal cortical extract should be given intravenously and 10 c cm intramuscularly together with 25 mgm of desoxycorticosterone acetate in oil intramuscularly. The intramuscular injection of cortical extract should be repeated six hourly during the first 24 hours. The intravenous injection of cortical extract has an immediate effect the intramuscular injection has its maximum effect three to six hours later and desoxycorticosterone acetate being more slowly absorbed produces a continuous effect for 24 hours.

Desoxycorticosterone which has been synthesised is but one

of the many compounds which have been isolated from the adrenal cortex. It is the most active in its ability to maintain life and regulates the electrolytic balance by decreasing the excretion of sodium and increasing the output of potassium. Since it is one of many and not the true adrenal hormone it is safer not to rely on this alone in the treatment of a condition so serious as a crisis but to give whole extract in addition.

During the second day of treatment the patient should be fed on fluids such as milk, chocolate and fruit juices and should be given a further two litres of the glucose saline solution intravenously. He should also receive 5 to 10 c.c.m. of cortical extract intramuscularly four to six hourly and one intramuscular injection of 10 to 15 mgm. of desoxycorticosterone acetate in oil. On the third day the injections of extract may be reduced to two separated by a twelve hour interval and 10 mgm. of desoxycorticosterone acetate should be given. If the patient is unable to tolerate fluids by mouth the intravenous glucose saline should be repeated. On the fourth day cortical extract may be discontinued if the patient's condition warrants it but the injection of 10 mgm. of desoxycorticosterone acetate should be repeated. The patient should now be started on 5 gm. of salt by mouth daily given in five divided doses dispensed in gelatin capsules but if he is unable to tolerate it it should be given intravenously in 1 per cent solution. No more than 5 gm. of added salt a day should be given when the patient is receiving desoxycorticosterone acetate in order to avoid œdema. On the fifth day he should be able to take a more solid diet which should be rich in carbohydrates and the dose of desoxycorticosterone acetate should be reduced to 5 mgm. daily.

Thereafter the patient has probably improved to such an extent that he may be treated as a chronic case of Addison's disease. The dose of desoxycorticosterone acetate may then be gradually reduced and the maintenance dose determined. The appearance of œdema due to excessive sodium retention indicates that the dose is too high throughout treatment the behaviour of the blood sodium should be observed.

Acute adrenal insufficiency is rare and the condition requires the therapeutic measures which have been described in the treatment of an Addisonian crisis. It may be caused by acute infections

thrombosis or bilateral hæmorrhage into the adrenals (Waterhouse Friderichsen syndrome). Its features are similar to those of a crisis, consisting of extreme asthenia and prostration vomiting, diarrhœa, abdominal pain dehydration, low blood pressure and coma. It may be accompanied by fever, and in the cases of adrenal hæmorrhage there will be local signs of abdominal distension and a palpable swelling in one or both of the renal regions.

Adrenal medullary tumour: Phæochromocytoma (hyperchromafinism).

This rare tumour which is usually non malignant, secretes adrenaline or some allied compound, and may be associated with paroxysmal or continuous hypertension. A hypertensive attack or crisis consists of sudden dyspnœa, tachycardia, headache, pallor, a feeling of constriction about the heart and epigastrium, and sometimes nausea and vomiting. There may be glycosuria, and pulmonary œdema may occur. Coma may result from hypertensive encephalopathy, and in some instances sudden death has been reported. A tumour may be palpable in the renal region but if not the case may be mistaken for one of hypertensive encephalopathy complicating essential hypertension. Pyelography may reveal displacement of the kidney by the tumour.

The emergency treatment for the condition is as for hypertensive encephalopathy, *i.e.* rest, a venesection of 15 fl oz of blood (*page* 421) and a lumbar puncture (*page* 374) to relieve the increased intra cranial pressure.

HYPOGLYCÆMIA

Although hypoglycæmia occurs most commonly during the treatment of diabetes mellitus (*page* 212) it may also arise in Addison's disease, Simmonds's disease, pituitary tumours, and myxœdema in all of which hormones which oppose the action of insulin are deficient. Severe anorexia and vomiting are partly responsible for the hypoglycæmia of an Addisonian crisis, and for the extreme hypoglycæmia which occurs as a terminal phase in Simmonds's disease. It has been observed in severe hepatic disease, *e.g.* cirrhosis where the storage of glycogen is impaired and in renal glycosuria, where there is an abnormal loss of glucose from the body. The secretion of excessive amounts of insulin

(hyperinsulinism) results from hyperplasia adenoma or more rarely carcinoma of the islets of Langerhans and gives rise to the rare condition known as spontaneous hypoglycæmia.

The early symptoms of hypoglycæmia are a sense of apprehension hunger pains sweating pallor weakness diplopia and tremor. When severe there may be inco-ordination of gait simulating drunkenness mental disturbances epileptiform convulsions and coma. The plantar response may be extensor in type. The convulsions must not be mistaken for idiopathic epilepsy as has occurred to the knowledge of the writer. The coma resembles normal sleep but has to be differentiated from other causes of coma. The finding of an abnormally low blood sugar will clinch the diagnosis but in the severe diabetic who may have had a high blood sugar for some time it may be within the normal limits.

If the attack is mild or moderate the only treatment necessary to cause the symptoms rapidly to abate is the giving of a few teaspoonfuls of sugar by mouth washed down by a cupful of water. If the patient is in coma or semi-coma 20 c cm of a 60 per cent solution of dextrose should be injected intravenously there is usually considerable improvement within 20 minutes. Some physicians prefer to give $\frac{1}{2}$ to 1 c cm of adrenaline hydrochloride or pituitrin subcutaneously. These hormones raise the blood sugar by converting liver glycogen into glucose and are usually effective in rousing the patient so that he can take sugar by mouth. They are ineffective in cases of hepatic disease where the stores of liver glycogen are depleted.

If the intravenous injection of glucose solution is rendered difficult by much muscular twitching the arm should be fixed in position by a splint fastened to the bed to facilitate the procedure. If he is having convulsions inhalation of chloroform may be necessary to enable the injections to be carried out or adrenaline hydrochloride should be given subcutaneously. When this has brought about improvement glucose solution may then be given intravenously or if he is able to swallow by mouth.

ALLAN W. SPENCE

CHAPTER XV

Medical Emergencies in Renal Disease

THERE has been so much confused thinking on the subject of renal disease and hypertension in the past that a few preliminary words on their relationship may be helpful. Goldblatt in 1934 showed that ischæmia of the kidneys artificially produced invariably gave rise to hypertension. Most renal diseases in man since they interfere with the circulation of the kidney are accompanied by ischæmia of that organ and in turn by a rise in the systemic blood pressure. This may be transient as in acute nephritis and pregnancy toxæmia or permanent as in the more chronic forms of renal disease. Whether so-called essential hypertension has its origin in the renal arteries cannot yet be finally decided but in any case it does not properly come within the scope of this chapter.

The important lesson to assimilate for our present purpose is that in any case of renal disease it is essential for clear thought and logical treatment especially when dealing with urgent symptoms to distinguish carefully between symptoms due to failure of the kidneys in their function as excretory organs and symptoms due to a rise in blood pressure with its inevitable effects on the heart and blood vessels.

Acute nephritis is a good exercise in this since its immediate dangers are more closely connected with the sudden development of hypertension than with renal failure.

ACUTE NEPHRITIS

In a sense acute nephritis even uncomplicated is always a medical emergency for it is clear to all who study this disease that it is in the first few weeks and probably only then that prompt and efficient treatment can affect the outcome. Far too frequently patients who show the initial signs of acute nephritis namely œdema hypertension and hæmaturia are sent on long bus journeys to wait in the draughty hall of an Out patient Department. To send them on such journeys when they should

be in bed between warm blankets is to commit a medical crime of the first order

Any patient at any age who shows these signs should be promptly put to bed and kept warm and if domiciliary treatment is not possible or desirable he should be transferred to hospital by ambulance

For the detailed treatment of acute nephritis reference can be made to standard text books but it may briefly be emphasised that rest in bed should be continued until all evidence of acute renal disturbance is over the œdema has disappeared and the blood pressure returned to normal. Diet for the first few days should consist of about a pint of fluid daily (preferably sweetened fruit juice) a few biscuits and nothing else

There are several ways in which the complications of acute nephritis may present as medical emergencies. Sometimes these complications are the first signs of disease more frequently they arise during the early days of a case already diagnosed

Heart failure in acute nephritis

Acute left ventricular failure not uncommonly results from the rapid development of hypertension

DIAGNOSIS—To the uninitiated and sometimes to the experienced this may present great difficulties in diagnosis. The patient is generally a child or a young adult who develops acute dyspnoea and orthopnoea which may be paroxysmal and nocturnal. There is cyanosis and congestive signs can be detected in the lungs. Occasionally there may be acute pulmonary œdema if the cardiac condition is not relieved. The right side of the heart may become involved as shown by distension of the neck veins and enlargement of the liver. The diagnosis rests on (1) the absence of the ordinary causes of heart failure in young persons (2) the presence of hypertension which in a young person immediately suggests acute nephritis and (3) red cells leucocytes and casts in the urine. Many of these patients have only slight œdema but if it is present in the face and hands a renal rather than a cardiac origin will of course be suggested. It should be remembered that albuminuria (without blood or cellular casts) may occur in any case of heart failure and that hypertension may diminish with the onset of left ventricular strain. In an adolescent with acute nephritis and

left ventricular failure the significance of a blood pressure of 150/100 should not be overlooked

Treatment—This is as in any other case of acute left ventricular failure with the exception that the mercurial diuretics cannot be used on account of the diseased kidneys Venesection (at least one pint in an adult) morphine and a restricted diet such as that outlined above are the three major therapeutic measures with of course complete rest In most cases improvement takes place in a few days and the prognosis is then that of an ordinary case of acute nephritis Occasionally heart failure is the complication determining a fatal outcome

Convulsions

It is established beyond doubt but not always fully appreciated that the *convulsions which occur in acute nephritis* (and also in eclampsia) are not due to uræmia There is still some controversy over their exact mechanism but they are undoubtedly related to sudden rises in blood pressure Oedema of the brain and arterial spasm are possible factors in their production The blood chemistry is often normal though in severe cases there may be varying degrees of nitrogen retention which bear no constant relationship to the liability to convulsions

Should convulsions be the first sign of the onset of acute nephritis the question of diagnosis again arises especially as a specimen of urine may be temporarily unobtainable There is nearly always some oedema of the hands and ankles and hypertension will be present The retina will rarely show any characteristic features but spasm of the arteries hæmorrhages and papilloedema occasionally occur A specimen of urine should be obtained (by catheter if necessary) as soon as possible and will show albumin blood and casts The presence of a trace of albumin *only* (i.e. without blood) after a convulsion would be more suggestive of epilepsy than of acute nephritis Although convulsions in acute nephritis add to the immediate seriousness of the case and may be a cause of death they do not add in any way to the gravity of the eventual prognosis in other words if the convulsions can be controlled there is no reason why the case should not progress satisfactorily to cure I have seen a number of patients who have had as many as 10 or even 20 convulsions in the early stages make a complete recovery

Treatment—There are several efficient methods of controlling the fits. Sedatives are valuable and as they are usually nearer to hand than is the apparatus for venesection and lumbar puncture it is usual to commence treatment by giving sodium phenobarbitone 3 grains in an adult intramuscularly. Alternatively paraldehyde may be given per rectum in doses of 0.5 c cm per kilo body weight in 10 times its volume of warm saline or in severe cases seven c cm of paraldehyde dissolved in warm sterile normal saline by slow intravenous injection.

Venesection is often successful. Lumbar puncture should be done with care using a manometer. If the pressure is high a few c cm of fluid should be removed and this may be repeated some hours later. Where there is gross oedema of the brain the sudden removal of a large quantity of fluid is probably as dangerous as it is in cerebral tumour.

If these measures prove unsuccessful the intravenous injection of hypertonic solutions will often succeed. Chlorides are to be avoided because of the renal condition and the best solution is 200 c cm of 50 per cent sucrose given *very slowly*. Another 100 c cm can be given some hours later if the convulsions recur. Another intravenous injection which serves a similar purpose is 20 c cm of 10 per cent magnesium sulphate administered at a rate of 2 c cm per minute. This may be given every two hours and if respiratory depression follows (which is the only likely untoward effect) intravenous calcium gluconate is the antidote. Magnesium sulphate (60 c cm of 50 per cent solution) may be given by the rectum every four hours if apparatus and solutions for intravenous work are not available and is also useful after the initial control of the fits by other measures.

The treatment outlined here for the convulsions of acute nephritis is equally applicable to any other form of what is collectively termed hypertensive encephalopathy such as eclampsia or the acute headache and vomiting sometimes accompanied by blindness convulsions and transient paralysis which may occur in any severe form of hypertension. In these latter cases the diagnosis from cerebral thrombosis and hæmorrhage is not always easy.

Oliguria and anuria

In some cases of acute nephritis the urinary secretion is

diminished to a few ounces only or even completely suppressed. This is liable to give rise to panic on the part of the medical attendant and it is common for a series of misdirected attempts at therapy to be applied in the hope of inducing the kidney to recommence secretion. Our knowledge of the changes in renal function which take place in acute nephritis is still incomplete but it seems certain that the glomerular circulation is considerably slowed down and that the resulting ischæmia of the kidney is the cause of the hypertension which is therefore to some extent compensatory. The administration of large quantities of fluid in acute nephritis is rarely followed by a corresponding increase in the amount of urine and may have the undesirable effect of merely increasing the œdema and the renal congestion and thus making matters worse. The proper course is to keep calm and reiterate the instructions that only a pint of fluid should be given in 24 hours. In nearly every case the kidney will recover spontaneously and the secretion of urine will be re-established in a few days.

Decapsulation of the kidney is a measure which has support in theory because it may relieve renal congestion. Many authorities especially in Germany teach that if anuria has existed for three days decapsulation should be carried out but the operation is not free from danger and a good many failures have been recorded as well as successes. Since most cases recover spontaneously one is always a little doubtful as to whether the success was actually due to the decapsulation. In short the operation is being employed less and less and will probably be abandoned altogether in the future.

(Suppression of urine due to causes other than acute nephritis is dealt with on page 237.)

We turn now to three conditions—uræmia, amblyopia and œdema which although they may occur in acute nephritis are more commonly the result of long standing renal disease. All may constitute emergencies under certain circumstances.

Uræmia

The term uræmia has been much abused in the past. It should be employed to indicate only a state of chemical poisoning caused by the retention of waste products in the blood. Thus

occasionally occurs in severe cases of acute nephritis and in such cases may be transient. Far more frequently it arises as the end result of chronic renal disease of all kinds including chronic nephritis malignant hypertension (nephrosclerosis) tuberculosis of the kidney chronic pyelonephritis polycystic kidney amyloid disease hydronephrosis enlarged prostate and in fact any condition leading to gradual destruction of the kidneys or progressive interference with their function. It has also been realised lately that severe trauma especially in the form of the crush syndrome may lead to rapid failure of renal function and to death from uræmia.

Consideration of this list of causes will reveal the fact that except in cases of prostatic disease uræmia is far more likely to occur in the young or middle aged and will enable us to avoid the mistake still far too common of confusing the results of hypertension in elderly people (such as cerebral thrombosis and hæmorrhage) with those of uræmia under the convenient but meaningless label of chronic interstitial nephritis. The diagnosis of uræmia in old persons is nearly always wrong.

Uræmia must also be distinguished from hypertensive encephalopathy as already stated. The first essential in the diagnosis of uræmia therefore is clear thinking. Uræmia being a state of chemical poisoning cannot give rise to such symptoms as hemiplegia or blindness despite what may still be read in some textbooks. Such symptoms may however co exist with uræmia as a result of an accompanying hypertension. The most common presenting symptoms of uræmia are anorexia and vomiting. These are accompanied by anæmia and loss of weight malaise weakness and headache. The tongue is furred and the skin dry. A history of polyuria attributable to renal failure can usually be elicited. Strange as it may seem all these symptoms may be overlooked or passed off as biliousness especially in cases where hypertension is not extreme. The first occasion on which the doctor is called may be when the terminal symptoms of uræmia namely drowsiness leading to coma muscular twitching convulsions severe dyspnœa or acute mania have suddenly developed. It is in this sense that uræmia becomes a medical emergency.

The absolute diagnosis of uræmia cannot be made until blood examination has revealed gross nitrogen retention (normal range

of blood urea and non protein nitrogen 20 to 40 mgm per cent)

Treatment —The treatment of uræmia depends on the cause. If there is urinary obstruction as in prostatic disease this must gradually be relieved in consultation with the surgeon. If the cause is acute nephritis the treatment is that of the underlying disease. In the far more common cases of uræmia due to advanced bilateral disease of the kidneys therapeutics is purely symptomatic. Methods of purifying the blood by dialysis and by peritoneal lavage although interesting experimentally have not yet proved sufficiently practical or lasting in their results to warrant general adoption.

Sedatives therefore although they may theoretically have an adverse effect on renal function should not be withheld in these hopeless cases. Phenobarbitone may control vomiting convulsions and excitement. True renal dyspnoea which is very distressing is caused by acidosis and may often be relieved by giving sodium bicarbonate in doses of 60 grains every two hours. For uræmic coma there is no effective treatment. Most patients with uræmia will not wish for more than a very light diet. If the underlying disease is very chronic and has hitherto received no adequate treatment improvement may occur on a diet restricted to biscuits fruit and vegetables with ample fluid (3 or 4 litres or about 6 to 8 pints per day). Milk is not suitable. There is no reason to restrict salt in the absence of oedema in fact salt reserves may have been much depleted by polyuria and vomiting thereby aggravating the uræmia. If blood tests and the patient's condition show that improvement has followed these dietary measures the protein intake should be increased to about 50 gm per day as a maintenance ration.

(Uræmia originating primarily in diseases outside the kidney is dealt with on page 241)

Amblyopia and blindness

Traditionally blindness when it occurs in kidney disease is referred to as amaurosis. This is mentioned not because the tradition is worth preserving but to emphasise the fact that the terms are synonymous. Amblyopia refers to a less complete affection which may be described in English as dimness of vision.

There are four mechanisms of visual disturbance in renal disease hypertension being the cause of each —

- (1) Hypertensive retinitis by causing œdema of the papilla retinal hæmorrhage and exudate
- (2) Thrombosis of retinal vessels
- (3) Detachment of the retina
- (4) Hypertensive encephalopathy. Here a circulatory disturbance of the visual cortex may lead to temporary blindness without retinal changes

If the cause of the hypertension is transient or curable as in pregnancy toxæmia acute nephritis and certain cases of unilateral renal disease the retinal lesions may heal but if they are far advanced permanent visual defects will remain

In amblyopia due to the retinitis of chronic hypertension temporary improvement may occur and it is worth while attempting to treat such cases if only to give comfort to the patient

Treatment—This can only be effective if applied to the hypertension. Complete and prolonged rest in bed on a light diet is indicated. A diet of low calorie value with restricted fluid is more important than the nature of the food itself. More drastic measures such as thoraco lumbar sympathectomy are not usually applicable to hypertension of renal origin except in some cases of persistent hypertension with good renal function following pregnancy toxæmia or pyelonephritis

Oedema

Oedema rarely gives rise to an emergency call except when it occurs in the glottis or the lungs or when hydrothorax and ascites are sufficiently extensive to cause respiratory embarrassment

Oedema of the lungs is almost invariably secondary to left ventricular failure the treatment of which has already been described. Fluid in the pleural and peritoneal spaces can be simply removed by paracentesis

Oedema of the glottis is a very rare but very dangerous complication of any form of nephritis accompanied by œdema. There is usually œdema of the palate and fauces at the same time and there may be pain in the throat dysphagia and aphonia before the acute symptoms of cyanosis and suffocation appear. The treatment is immediate tracheotomy (*see page 272*)

URINARY INFECTIONS

It is only rarely that acute pyelonephritis can be called an emergency but occasional cases with severe general symptoms require urgent relief. In this type rigors, pyrexia (often 103°F) and intense headache are usually the prominent features, the local symptoms such as pain in the back, frequency and dysuria being much less in evidence. The differential diagnosis from meningitis may even arise. On the other hand dysuria itself may be so distressing as to require immediate treatment.

Diagnosis—In either case the provisional diagnosis can usually be made in a few minutes by placing a drop of the cloudy urine on a glass slide, covering it with a coverglass and examining it microscopically with a dry lens. Pus cells and bacteria in large numbers are immediately apparent. The diagnosis should of course be confirmed later and the nature of the causative organism determined by the bacteriological examination of a specimen collected aseptically.

Treatment—Despite the efficacy of the newer methods of treatment of urinary infections, namely mandelic acid and the sulphonamides, the quickest control of symptoms is still to be obtained by the administration of large doses of alkali.

At least 4 gm (60 gr) of sodium bicarbonate and 4 gm of potassium or sodium citrate should be given every two hours until the temperature is subsiding and the urine is strongly alkaline to litmus. The dosage may then be reduced. There is no special merit in the mixture except that the citrates give rise to much less gas in the stomach than do the bicarbonates. If vomiting is troublesome the medicine may have to be given in quarter doses at half hourly intervals. At the same time providing that vomiting permits large quantities of fluids should be given, i.e. at least 4 litres per day.

Alkalis, although completely inhibiting the action of the mandelates, do not interfere with the antibacterial action of the sulphonamides and 1 gm of sulphathiazole may be given four hourly at the same time. If there is any tendency to vomiting however it is best to give alkalis first alone and add sulphonamides when the acute symptoms have passed. Within a few days bacteriological reports will be at hand to determine the further conduct of the case. It will be remembered that acute

pyelonephritis may complicate renal calculus and other disorders of the urinary tract and not infrequently occurs during pregnancy. Pregnancy is not a contra indication to the treatment outlined above.

We may now turn to a brief consideration of three major symptoms of renal disorder which may be caused by a variety of conditions namely renal colic hæmaturia and suppression of urine. Obviously the essential in each case is to make an accurate diagnosis but full consideration on these lines would be beyond the scope of this chapter. Nevertheless as the symptoms themselves may constitute a medical emergency their immediate treatment requires discussion.

RENAL COLIC

The only difficulty about renal colic as an emergency is to be reasonably sure of the diagnosis. If the pain is severe morphine will be required. This is of course a dangerous drug to give if the case should after all turn out to be one of appendicitis perforation or intestinal obstruction for morphine may obscure the signs and symptoms and dangerously delay the necessary intervention of the surgeon.

We cannot here discuss the diagnosis in detail. The important points to remember are the characteristics of renal colic its sudden onset and spasmodic nature the fact that the patient doubles up or rolls about in the spasms and the absence of rigidity or muscle guarding. The distribution of the pain may not be characteristic but in typical cases it strikes from the loin to the groin and often down to the testicle and inner aspect of the thigh. In severe colic cold sweats and vomiting are usual. A history of previous similar attacks may be helpful as will a negative history of gastro-intestinal disorder.

The differentiation from biliary colic may be difficult also but is of less immediate importance since morphine (or better Dilaudid gr $\frac{1}{32}$) is indicated in both conditions. Mild attacks of renal colic are common in the tropics where excessive sweating leads to dehydration and urinary concentration.

Treatment—The treatment of the less severe cases is by atropine rest and the application of heat to the loin. In severe cases morphine is required. When the attack has subsided an

accurate diagnosis as to causation must be made, and will probably necessitate admission to hospital for radiological and cystoscopic examinations

HÆMATURIA

Hæmaturia, although alarming to the patient, never presents a medical emergency in the sense of being in itself dangerous to life. Treatment therefore consists in keeping oneself and the patient calm until such time as the cause can be fully investigated. The value of cystoscopy at the time of hæmaturia should not be overlooked if circumstances permit of this.

So many of the causes of hæmaturia are serious ones (e.g. papilloma of the bladder and renal tuberculosis) that to omit thorough investigation or to postpone it until some further symptoms develop, constitutes serious negligence. This applies even though the hæmaturia ceases spontaneously (as it usually does) in a few days.

Occasionally the passage of clots down the ureter gives rise to colic and even to obstruction which will require appropriate treatment including surgical collaboration.

ANURIA. (SUPPRESSION OF URINE)

Anuria, or failure of the secretion of urine, must always be distinguished from retention of urine. In the latter case the bladder will be full in the former empty.

Apart from the anuria which occasionally occurs in acute nephritis, to which reference has already been made, anuria may occur at the termination of any destructive bilateral disease of the kidneys.

Anuria may also be found in the following conditions —

- (1) Poisoning by mercurial salts and occasionally by other substances
- (2) Urinary obstruction, for instance bilateral renal calculus
- (3) Sulphonamide overdosage
- (4) Renal anoxia
- (5) After operations on the urinary tract (reflex anuria)

As the causes are so diverse there is usually not much difficulty in making the diagnosis.

Treatment — This depends on the cause.

POISONING BY MERCURIAL SALTS—Several raw eggs in milk should be given immediately to precipitate any mercury in the stomach which should then be thoroughly washed out

Sodium formaldehyde sulphonylate (British Drug Houses) which reduces mercuric chloride to the mercurous form is then used in 5 per cent solution to wash out the stomach and 200 c cm of the solution are left in. The same volume of solution may then be given slowly by intravenous drip and repeated after six hours. Later treatment is designed to combat the salt and water depletion resulting from diarrhoea and consists of continuous infusion of intravenous saline to which glucose can be added. Decapsulation of the kidney is probably useless.

The most recent treatment is by BAL (British Anti Lewisite) (Boots) a substance known chemically as 2,3-Dimercaptopropanol which was elaborated to protect against arsenical poisoning by lewisite. Heavy metals are toxic to biological systems because of their reaction with SH groups of the protein moiety of cellular enzymes to form mercaptides. Mercury shares this action. BAL is capable of reactivating enzyme systems poisoned by mercury.

Longcope and Luetscher* have recorded the results of treatment in 23 cases of poisoning by mercury bichloride. All recovered except one, an early case in which inadequate doses were given. Nine of the patients had swallowed from 1.5 to 20 gm amounts which are likely to cause death in a high proportion of cases. The authors recommend the following treatment—

- (1) The stomach is washed out with 5 or 10 per cent sodium formaldehyde sulphonylate and 300 mgm of BAL (10 per cent solution in benzyl benzoate and peanut oil) is injected intramuscularly.
- (2) One or two hours later 150 mgm BAL are given followed by 150 mgm in four to six hours.
- (3) Another dose may be given within 12 hours in severe cases and thereafter two doses per day for the next two or three days depending upon the patient's condition.
- (4) Dehydration and shock are treated by intravenous infusion of saline and glucose and by blood transfusion as necessary.

* LONGCOPE W. T. and LUETSCHER J. A.—*J. Clin. Invest.* 1946 25, 557

REFLEX ANURIA—This is usually transient and is treated by the administration of copious fluids by mouth and isotonic saline intravenously

SULPHONAMIDE ANURIA—This is a very important condition now that sulphonamide drugs are in such constant use. Fortunately it is rare but it is a most serious emergency since neglect of prompt and effective treatment may be fatal

Certain members of the sulphonamide series especially sulphapyridine, sulphathiazole and sulphadiazine are excreted as relatively insoluble acetyl derivatives and in certain circumstances depending on the reaction and concentration of the urine crystals of the acetyl salts are deposited in the renal tubules. The whole renal pelvis and ureter may become blocked by a mass of crystals, blood and debris. This may occur at any stage but always indicates failure to observe proper precautions during sulphonamide treatment. If sufficient fluid and alkali are given sulphonamide anuria cannot occur but it must be remembered that patients on sulphonamide treatment are often dehydrated by sweating, pyrexia and vomiting and therefore an abnormally large quantity of fluid may be necessary in order to ensure an adequate urinary flow. Moreover sickly patients will not always willingly take enough fluid and the patient's attendants may not be sufficiently diligent in seeing that fluid ordered is actually consumed.

SYMPTOMS—As a rule the first symptom is renal and ureteric pain which may be severe. A small quantity of dark chocolate coloured urine is usually voided after which in the worse cases the flow of urine ceases altogether.

Treatment—Action must be immediate. Fluids by mouth in large quantities must be given along with alkali as in the treatment of urinary infections described above. If urinary secretion is not re-established within 12 hours or severe oliguria persists after 24 hours cystoscopy should be performed, ureteric catheters passed and the renal pelvis washed out with sodium bicarbonate solution (2.5 per cent). The sulphonamide should of course be stopped at once but if the infection for which it was being given persists *sulphanilamide* can be administered safely if appropriate since it does not form an insoluble acetyl salt.

NOTE ON THE USELESS TREATMENTS OF ANURIA

It has been the custom in the past in cases of anuria however caused to give intravenous sodium sulphate because of its action as a diuretic. Sulphates act as diuretics only by virtue of being non threshold substances. In other words they are not re absorbed in the tubules and so exert an osmotic action interfering with the re absorption of water. This treatment is unphysiological. Sulphates cannot act in restoring the renal circulation any better than saline solution. If it is possible to restore the renal circulation the secretion of urine will automatically recommence. The addition of sulphates only robs the body of fluid which it can ill afford.

Mercurial diuretics have no place in the treatment of anuria caused by renal failure. Their value lies in the removal of excess of fluid (œdema) from the body in cases such as heart failure where the kidney is still capable of active secretion.

RENAL ANOXIA AND THE CRUSH SYNDROME

Renal anoxia is a term recently introduced by Macgrath and his co workers and requires some amplification. For many years it has been known that anuria may occur in conditions accompanied by shock and peripheral vascular failure. In patients who recover the anuria is followed by a period of partial renal failure with polyuria and azotemia. Similar cases have been described as *traumatic uræmia in severe battle casualties not affecting the kidneys directly*. Pathologically the main lesion is degeneration of the tubules.

In air raid casualties where a limb has been pinned down for some hours a syndrome of renal failure may develop after release leading to death from uræmia. Finally in conditions such as blackwater fever and mis matched blood transfusion a similar syndrome may develop and in diseases associated with severe dehydration such as cholera and infantile gastro enteritis renal failure with anuria may terminate the illness.

Probably there are several factors at work such as dehydration and damage to the tubules by the excretion of pigment in some instances. In the crush syndrome myohæmoglobin from the injured muscle is found in the renal tubules. But diminished renal blood flow with consequent anoxia appears to be a factor common to all these conditions.

Treatment—This consists in combating the peripheral circulatory failure by all appropriate methods—in shock—plasma in hæmorrhage—blood transfusion and in dehydration—intra venous saline. In cases where pigment (hæmoglobin or myo hæmoglobin) is being excreted it is an advantage to give alkali in addition since deposition of pigment in the renal tubules occurs more readily in an acid medium. Sodium rather than potassium salts (citrate and bicarbonate) should be used because a large amount of potassium is absorbed from injured muscle and may be a factor in the causation of the crush syndrome. In short these cases of anuria and renal failure require prompt and energetic treatment directed to restoring the renal circulation.

EXTRA RENAL URÆMIA AND ALKALOSIS

The term extra renal uræmia has been applied to a number of conditions some of which should probably be included under renal anoxia. Thus partial failure of renal function occurs in severe hæmorrhage (*e.g.* hæmatemesis and melæna) in cases of chloride deficiency (*e.g.* vomiting) and also in alkalosis.

The uræmia of alkalosis

This is worth describing separately because it may constitute an emergency in the sense that prompt and appropriate treatment may be necessary to avert death.

It is less common now than formerly when it was chiefly seen in patients suffering from gastric and duodenal ulcer receiving treatment with sodium bicarbonate. Weakness headache nausea vomiting and drowsiness are its chief symptoms. Tetany may occur and albuminuria is sometimes present. The blood urea may reach 100 mgm or more per 100 c cm. In cases of pyloric stenosis loss of chloride by vomiting may aggravate or initiate the condition. There is a danger that the condition may pass *unrecognised and the symptoms be treated by still more frequent doses of the alkaline powder which is causing the syndrome*.

Treatment—Stop all alkali immediately and give copious fluid. The symptoms will then gradually subside. More urgent symptoms necessitate the intravenous injection of calcium gluconate *e.g.* 10 c cm of 10 per cent solution followed by continuous intravenous infusion of isotonic saline. If an antacid

is still required magnesium trisilicate may be substituted. If pyloric stenosis is present operation will be necessary.

CONCLUSION.—As knowledge advances we become better equipped to think in physiological terms about disorders of the kidneys and the symptoms they produce. We can separate the mechanical from the functional disorders and the syndrome of hypertension from that of uræmia even though they co-exist. We can distinguish between changes which are reversible and those which are the end results of destructive disease and a more and more conditions become amenable to treatment there is less and less excuse for mischievous interference with those which are incurable. The mild diaphoretic mixture with its four or five ingredients is just as dead as the drastic purge, the pilocarpine injection and the hot air bath.

ROBERT PLATT

CHAPTER XVI

Medical Emergencies in Infancy and Childhood

IN this chapter the general and nutritional aspects of medical emergencies in infancy and childhood will be considered first. Special emergencies will then be described system by system.

NUTRITIONAL FAILURE

Perhaps the most important group of medical emergencies in childhood is that which includes all types of rapid nutritional failure, with which are frequently associated various dangerous forms of metabolic disturbance. Many of the patients are infants; the younger the child, the more rapidly does the disorder progress and the more serious is the prognosis.

Several clinical varieties occur—

- (1) Dehydration (or anhydraemia) as in gastro-enteritis, pyloric and intestinal obstruction, and all other diseases causing vomiting or diarrhoea, *e.g.* cyclical vomiting.
- (2) Wasting of body tissues, as in starvation and other dietetic errors, coeliac disease, septicæmia and other acute infections and advanced tuberculosis.
- (3) "Surgical" shock resulting from trauma, hæmorrhage, burns and scalds, intussusception, volvulus and other types of intestinal obstruction.
- (4) Acidosis, ketosis and alkalosis.

In many cases these manifestations overlap, no matter how clearly defined and limited a metabolic disorder may be at its beginning; secondary effects are liable to develop rapidly. Some of these may be compensatory readjustments and while treatment should be as comprehensive as seems necessary after a careful assessment of a particular situation, care must be taken to avoid interference with spontaneous physiological responses which are likely to be helpful to the patient.

By taking precautionary measures early in the course of the illness it may be possible to avoid the development of a critical

normal saline or half strength Ringer lactate solution* but a high percentage of sugar (e.g. 10 per cent dextrimaltose in water and flavoured with fruit juice) may be given by mouth to a patient who is not suffering from diarrhoea or vomiting. On the other hand it is wise to omit the carbohydrate during the early treatment of infantile gastro-enteritis because it may give rise to flatulence from intestinal fermentation. In such a case a little saccharin may be used if necessary to sweeten the fluid. As the child recovers a gradual change may be made to human milk if available or to a half cream milk mixture or lactic acid milk.

Older children of highly strung type suffering from cyclical vomiting should at first be offered quite small volumes by mouth e.g. one dessertspoonful hourly for three hours then one tablespoonful hourly with steady increase. It should be administered by a sympathetic but firm individual (preferably not a relative because the psychological aspect is important).

Parenteral routes of administration and types of fluid

When it is necessary to give fluid parenterally as in a very acute emergency or when there is vomiting one has the choice of the subcutaneous the intramuscular the intravenous and the intraosseous routes. In addition to the technical difficulties of administration and the possibility of introducing infection these methods may present certain other disadvantages such as rapid diuresis after intravenous administration and delayed absorption from subcutaneous and intramuscular injections. Furthermore acute cardiac embarrassment may result if fluid is transfused too rapidly into the circulation. Generally speaking the most effective method of parenteral administration is by intravenous transfusion and 5 per cent dextrose in half strength normal saline is a suitable fluid. Sometimes when there has been loss of salt by vomiting it is well to start with 5 per cent dextrose in either normal saline or Hartmann's solution and after about one fifth of the total fluid requirement for the day has been administered to continue with 5 per cent dextrose in half strength normal saline.

*Liquor Ringer Lactatis as described on page 17 of the 1941 Supplement to the British Pharmaceutical Codex of 1934. Synonym Hartmann's solution by which name it will be referred to here. Some physicians prefer to add to this solution Magnesium Chloride 0.02 per cent.

(or half strength Hartmann's solution) During the first two hours the injection may be given at the rate of 20 drops per minute (about 70 to 80 c cm per hour) but subsequently the rate of flow should be reduced to 10 drops per minute (about 35 to 40 c cm per hour) It is seldom wise to give more than 20 fl oz (600 c cm) in 24 hours intravenously to an infant under six months of age and when bronchitis or any other respiratory disease is present special care must be taken to avoid giving too large a volume of fluid

When dehydration is very severe blood plasma (10 c cm per pound of expected body weight) should be transfused quickly e.g. in a period of about 20 minutes to be followed by saline as recommended above

When the immediate danger of dehydration has been removed usually after 24 hours of intravenous infusion human plasma may again be given with advantage It is added to the infusion fluid in the proportion of 1 part to 3 or 4 until oral feeding is established When there is obvious anaemia a transfusion of compatible blood preferably fresh may be given the total volume administered to infants being 10 to 15 c cm per pound of body weight

For subcutaneous and intramuscular injections normal saline or Hartmann's solution is generally used either as single injections of 50 to 100 c cm six hourly or by drip technique at the rate of 3 to 10 drops per minute (about 12 to 40 c cm per hour)

The same fluids may be used for intraosseous injection but sometimes the rate of flow is very slow at first and it is rather difficult to control This method would appear to be of special value when venous spasm prevents successful intravenous infusion but there is some danger of causing osteomyelitis

If a stimulant is needed injection of nikethamide 0.5 c cm may be given intramuscularly every two hours for 4 to 6 doses

WASTING OF TISSUES

There is a decrease of blood protein in most cases of starvation whether from lack of food or diseases such as septicaemia advanced tuberculosis coeliac disease severe diarrhoea excessive albuminuria many diseases of the liver and extensive burns Although a patient cannot be adequately nourished by the parenteral administration of food for more than a short period

many children have been successfully tided over a dangerous crisis by this treatment. Frequently in such cases the child needs preliminary treatment for dehydration subsequently dried plasma recombined with sterile water in three or four times its normal concentration may be given by intravenous drip or fresh plasma or serum may be used. Not more than 15 to 25 c cm of blood plasma per pound of body weight can be administered with safety in 24 hours.

If it is decided to give a mixture of amino acids and polypeptides (derived from casein or meat proteins by enzymic digestion) an infusion of protein hydrolysate (*e.g.* Casydrol) may be given at a rate not exceeding 50 c cm per hour. The daily protein requirement for children under six years is 3 gm per kg of body weight (*i.e.* three times the requirement of an adult). This is equivalent to about 25 c cm of Casydrol solution per pound. Twenty five gm of glucose should be given at the same time—either by mouth or parenterally—and vitamin B in a dosage of 600 international units daily by mouth.

SURGICAL SHOCK

No time should be lost in providing adequate warmth to the body and extremities and oxygen by inhalation. Blood transfusion is usually an urgent requirement and it may need to be repeated so long as the patient's systolic blood pressure remains low say below 75 mm Hg in a child of six years. In some cases for instance after burns and scalds when there has been considerable loss of protein it may be necessary to infuse blood plasma over prolonged periods.

ACIDOSIS KETOSIS AND ALKALOSIS

Dangerous fluctuations of the blood pH occur fairly frequently in childhood particularly in infancy and in many cases dehydration is an accompaniment. Clearly an attempt should be made to deal as speedily as possible with the primary cause but *valuable and often life-saving assistance may be rendered by effective treatment of dehydration.* Hartmann's solution is suitable for infusion when acidosis or ketosis is present and sodium bicarbonate 15 gr four hourly by mouth in mixture form may be given to a child of one year and will be helpful. For the

infant suffering from both diarrhoea and vomiting Hartmann's solution is the most suitable for infusion. On the other hand when alkalosis is present *e.g.* in congenital pyloric stenosis or high intestinal obstruction the loss of chloride may best be made good by infusion of normal saline solution. Tetany may be a complication (*see page 241*)

ALIMENTARY SYSTEM

The correct diagnosis of a sudden abdominal disorder in childhood—a frequent problem for the medical practitioner to solve—calls for considerable care and it will not be out of place to make some comments upon the technique of examination and the significance of certain symptoms and signs. First and foremost must be stressed the importance of obtaining a detailed history to do this takes time but lessens the likelihood of a mistake. It is necessary to know whether the child has been in full health up to the onset of the present illness and whether indigestible or unusual food has been eaten recently what has been the sequence of the various symptoms whether there has been pyrexia and whether any symptoms perhaps nothing more than a slight cough have occurred to suggest a supra diaphragmatic cause of the illness. Before the doctor begins his examination he should have obtained so detailed a history that he can visualise quite clearly the successive events which have marked each day. This demands close attention to the mother's story and steady mental concentration. For instance merely to ask

Has he been sick? is not enough one wants to know. When did he first vomit? How many times has he vomited?

How was it related to the abdominal pain? and What has been vomited?

While it is true to say that many examples of abdominal disorder are caused by relatively mild illness such as simple indigestion each one may represent the beginning of a serious or potentially serious illness such as peritonitis appendicitis tuberculosis intussusception volvulus pneumonia purpura and visceral bleeding. Therefore even when the reported facts point to the likelihood of some such trivial disorder as an acute gastric upset following dietetic indiscretion the possibility of one of the

serious states must still receive full consideration. Extra abdominal illness such as tonsillitis, rheumatic infection and pneumonia especially when accompanied by pyrexia is more likely to cause abdominal pain and an occasional vomit in childhood than in adult life. Hence the routine examination of the child must include a view of the tongue, mouth, throat and ear drums, the taking of the temperature, examination of the heart, lungs, nervous system and urine. Naturally most attention will be devoted to the abdomen and examination of this must be carefully and sympathetically made. If the doctor is not known to the child some time should be spent in making friends. Then the routine examination of the abdomen (inspection, palpation, percussion and sometimes auscultation) is carried out.

Severe abdominal pain in a child is commonly of colicky nature and infants react to it by screaming and drawing the knees up on to the abdomen. Vomiting and distension are other urgent features which may point to acute abdominal disease; they demand a quick decision regarding the possible need for surgical intervention. Little help can be expected from the child himself who will have difficulty in describing and locating his pain, but valuable indications may be noted if his behaviour is closely watched. In addition to a careful abdominal and general examination the rectum should invariably be examined also. A leucocyte count often helps to differentiate inflammation from other states.

VOMITING AND DIARRHŒA IN INFANCY

This syndrome—one of the commonest disorders in infancy—occurs sporadically and may then be mild and readily amenable to treatment, but it also occurs in epidemics and may then be of great severity with a mortality approaching 90 per cent. The degree of dehydration largely determines the severity of the illness; in the worst cases the child's weight falls rapidly despite the administration of parenteral fluid and other forms of treatment until at length irreversible tissue changes occur, notably in the liver, and recovery is then unlikely.

The illness occurs mainly in artificially fed children and there is good reason to believe that it would practically disappear if breast feeding were more widely practised. Obviously prevention is the ideal, but if that is not possible it is important to identify

and remove the cause quickly. Both grossly unsuitable feeding and under nutrition, especially when due to the use of a very dilute sweetened condensed milk mixture, might be expected to lower the resistance of the alimentary tract to infection, but it is not so generally realised that overfeeding may have the same effect. This may occur even in breast fed babies and can be confirmed by test weighing. Rapid gain of weight, vomiting, and the passage of curds with subsequent loss of weight should suggest the likelihood of overfeeding. If the baby is being fed on whole cow's milk, it may be found that only the milk from the upper portion of the vessel, containing most of the cream is being used. The fat of cow's milk is more likely to cause indigestion than the other constituents, and may thus be responsible for the diarrhoea and vomiting.

To what extent diarrhoea and vomiting may be due to infection, as distinct from unwise feeding, is difficult to determine in any particular instance, but it is obvious that the more serious cases are infective in nature because the illness may spread to other babies, especially in hospitals and nurseries. In such circumstances tragic emphasis may be given to the truism that babies are generally best cared for by their mothers in their own homes. Sometimes the infection is contracted from an adult such as a nurse who has had an attack of diarrhoea and in these cases it may be possible to isolate Sonne or Flexner bacilli of dysentery. The infected adult may be suffering from a common cold rather than an alimentary disease, or pyoderma in the mother may result in severe gastro enteritis (staphylococcal) in the child. Possibly the presence of staphylococci in the mother's milk may be responsible for those rare examples of gastro enteritis in breast fed babies.

Generally it is assumed that an infection causing diarrhoea and vomiting produces inflammatory changes in the wall of the intestine but this is not necessarily so for these changes may be conspicuously absent at autopsy. This is a reminder that a diligent search should be made for signs of infection elsewhere. For instance, diarrhoea and vomiting may be the first indication of the presence of otitis media or pyelitis, the former is of special importance and the ear drums should be repeatedly inspected in all babies suffering from gastro-intestinal disorder. Pulmonary

disease may also be responsible though certainly less often than middle ear inflammation

Management of vomiting and diarrhoea.

In a disease in which the condition of the child may undergo marked fluctuations from hour to hour it is impossible to lay down a regime suitable for all cases and much resource is needed in the treatment of urgent symptoms as they arise. It must be emphasised that skilful and devoted nursing is the keystone of treatment. The following schedule will serve as a basis for the handling of most cases

1 If the patient is seen early in the illness and there is a probability that the disorder is due to unsuitable diet a small dose of castor oil (60 m for a child under one year 120 m if over one year) may be helpful but it should not be used if there is any suspicion of acute appendicitis or peritonitis or of any type of intestinal obstruction

2 Milk feeds should be discontinued in favour of two to three hourly feeds of half strength Hartmann's solution (sweetened with saccharine not glucose). The total fluid intake should be $2\frac{1}{2}$ fl oz per pound in 24 hours the calculation being made on actual weight at first and later as the child begins to improve on 'expected' weight. A volume not exceeding 10 fl oz daily should be added to offset dehydration

3 When dehydration is serious fluid should be given intravenously (*Batman needle* page 427) or into the bone marrow (*Behr needle* page 438). Half strength Hartmann's solution containing 5 per cent glucose is suitable. After 12 to 24 hours of this treatment human plasma (1 in 4) may be added to the transfused fluid

4 At the end of 24 to 48 hours of treatment it may be possible to give half cream dried milk diluted with Hartmann's solution three hourly as follows—

(a) For 12 hours one part milk (reconstituted) to eight parts of Hartmann's solution

(b) For the next 12 hours two parts milk (reconstituted) to seven parts of Hartmann's solution with progressive increase until full-strength half-cream milk is reached

5 Drugs—phthalyl sulphathiazole 0.125 gm per lb in 24 hours

Injection of nikethamide 0.5 c cm intramuscularly two hourly for collapse

It will be necessary to increase the feeds more slowly in some cases and the need to rest the alimentary tract for a sufficient period requires emphasis. Nothing but harm can result from resuming even modified milk feeding too soon and as long as 72 hours on Hartmann's solution only whether by mouth or parenterally may not be excessive. Full cream milk should not be given for some weeks following recovery for the intestine tends to be intolerant of fat after an attack of gastro enteritis. Drugs are of secondary importance in this disease most of the sulphonamide group and penicillin have alike proved disappointing and their chief use is in the treatment of parenteral infections. Kaolin is without effect. Phthalyl sulphathiazole is most likely to be efficacious when the infecting agent is a dysentery bacillus. Administration of the fat soluble and other vitamins may have to be suspended while acute symptoms are still present.

OTHER URGENT ABDOMINAL CONDITIONS

Intussusception tends to occur in late infancy and well made boys are chiefly affected. There are sudden spasms of colicky pain which cause screaming and are followed by the passage of a stool. The baby is quiet and apparently normal between the attacks but as the condition advances the stools are found to consist mainly of blood stained mucus. Examination may show an abnormal emptiness in the right iliac fossa (signe de Dance) and a tumour consisting of the intussuscepted ileum and caecum may be felt in the upper part of the abdomen or on the left side. The apex of the mass can sometimes be felt in the rectum. Vomiting distension and other symptoms of intestinal obstruction occur late and it is characteristic of the illness that constitutional disturbance is seldom noteworthy in the earlier stages. Thereafter rapid deterioration occurs leaving the child in poor condition for a severe abdominal operation such as extensive resection of the bowel.

These conspicuous features of intussusception are described because it is important to differentiate the condition from acute ileo colitis (*page 41*) and from Henoch's purpura (*page 142*)

Acute appendicitis (*see also page 81*) is no less common in children than in adults. The clinical features are similar to those met with in older subjects and difficulty arises mainly in a young child who is unable to describe the pain he experiences or in a child whose appendix is abnormally situated. In the latter case confusion is likely to arise with certain other acute abdominal conditions more or less peculiar to childhood. **Lymphadenitis** (*page 39*) in the ileo-cæcal angle may result in pain in the right iliac fossa with slight pyrexia. A tender swelling is palpable but there is no distension or rigidity and constipation is not a feature. **Pyelitis** may give rise to pain in the right side of the abdomen; this diagnosis is suggested when pus cells are found in the urine. A right sided **perinephric abscess** may be suspected when the inflamed appendix is behind the cæcum but an appendix abscess is usually at a lower level than the kidney and seldom points posteriorly. **Basal pleurisy** (*page 44*) on the right side frequently gives rise to pain in the right iliac fossa and the signs of pneumonia with which it may be associated may not appear until later. The respiratory rate is however generally raised, rigidity is not marked and no local swelling can be discovered.

Pneumococcal peritonitis, which may closely resemble appendicitis is differentiated from the other forms of acute peritonitis by certain special features. It is rarely seen except in childhood and is much commoner in girls than in boys. The majority of cases are primary and are believed to arise from an ascending infection via the genital tract; less frequently it is secondary to pneumococcal infection elsewhere usually in the chest. The onset may be somewhat insidious with lower abdominal pain. The accompanying diarrhoea serves to distinguish the disease from peritonitis of other types for in these constipation is invariably present. There is rigidity of the abdominal wall and vomiting and distension slowly develop. The face may be flushed and herpes is not uncommon. In doubtful cases peritoneal aspiration is a helpful diagnostic measure. Treatment is by sulphadiazine and penicillin. In patients who have come under observation in a late stage one or more loculi of pus may be present and may require surgical drainage. Operation should be deferred until time has been allowed for the abscess to become well separated by adhesions from the rest of the peritoneal cavity.

RESPIRATORY SYSTEM

Cyanosis and dyspnœa of abrupt onset are symptoms which bring certain diseases of the respiratory system within the category of medical emergencies. To these may be added toxæmia the result of pneumonia or other acute infective process in the lungs or pleuræ whereby the threat to life is further increased.

Asphyxia neonatorum

Cyanosis with respiratory depression is the main feature of asphyxia livida its cause is usually to be sought in obstruction of the placental circulation during birth or of the respiratory passages after delivery. If unrelieved cyanosis gives place to pallor (asphyxia pallida) as the circulation fails. White asphyxia *à l'initio* suggests depression of the medullary centres by drugs or anæsthetics by intra-cranial injury or by maternal toxæmia. Thus prolonged and difficult labour, premature rupture of the membranes, prolapse of the cord, breech pre-presentation and the various obstetrical manipulations necessitated by these conditions predispose to asphyxia of the newborn infant.

The possible effect upon the child of every measure designed for the relief of the mother should be carefully weighed. In breech deliveries the body of the infant must be kept warm pending the delivery of the head, otherwise premature inspiration may occur with aspiration of fluid from the birth canal. If there is cyanosis the child should be inverted and suction by a small catheter used to remove fluid from the hypopharynx and trachea. Gentle rhythmic compression of the chest wall may then be sufficient to establish normal respiration. If this fails the infant should be immersed in a bath of water at 100°F and artificial respiration continued. Oxygen with 7 per cent carbon dioxide is given if recovery is delayed it can be administered by nasal catheter or by a soft rubber funnel *closely applied*. Later an oxygen tent may be used. Nikethamide injection 0.5 c cm is one of the best stimulants to employ and can be repeated half hourly, alternatively lobeline may be injected in doses of 1.5 mgm and in very urgent cases the umbilical vein may be used as the channel of administration.

In carrying out all these procedures it is of paramount importance to be very gentle for the condition of the infant is analogous to that encountered in surgical shock. Warmth

is essential and when breathing is established the infant is placed in a crib heated by suitably protected hot water bottles or an electric blanket. Careful observation is necessary even after improvement has set in for relapse is always possible. In cases where cerebral damage is thought to have occurred—and this will be suggested if there are convulsions localised twitchings or a tense fontanelle—60 grains of magnesium sulphate dissolved in an ounce of water may be injected into the rectum to reduce intracranial pressure. Lumbar puncture while useful for diagnosis has no other advantage and may do harm.

Certain congenital anomalies produce clinical signs closely resembling those of asphyxia neonatorum for instance congenital cardiac deformity tumours of the thymus malformation of the respiratory passages and diaphragmatic hernia. The outlook varies with the severity and nature of the lesion and the emergency treatment must be on the lines already recommended for asphyxia.

Pneumonia

Though lobar pneumonia is hardly less common in childhood than bronchopneumonia the latter is more serious and will therefore receive special consideration in this section. It occurs with particular frequency in states of debility such as those produced by rickets and chronic nutritional disorders. Upper respiratory catarrh and bronchitis are frequent precursors. A variety of organisms may be responsible including pneumococci streptococci staphylococci micrococcus catarrhalis and hæmophilus influenzae. Since their sensitivity to penicillin and sulpho namides is variable it is advantageous to test this when facilities are available. Bronchopneumonia may be the earliest manifestation of influenza measles or whooping cough and the possibility that the child may be suffering from an infectious disease should not be forgotten. Infants should be nursed in a well ventilated room kept at an even temperature of about 65°F but older children may be nursed with advantage in the open air. To facilitate easy breathing the patient should be propped up with pillows. No restraint should be placed on the chest by heavy clothing or tight bands and these points should be particularly borne in mind if a poultice or a gamgee jacket is ordered. Glucose drinks should be given liberally the water aids the

elimination of toxic products while the glucose nourishes the myocardium. A drug of the sulphonamide series should be given in every case, *e.g.*, sulphadiazine 0.25 gm. for an infant under six months or 0.5 gm. for an older child, administered four hourly, day and night until the temperature has been normal for 48 hours. If the child's condition is not improving after 24 hours, penicillin should be given in addition a suitable dosage is 10,000 units in one c.c.m. given by intramuscular injection three-hourly, and it can be reduced gradually when the pyrexia has permanently subsided (*see also page 442*). It is better to use a sulphonamide drug than to rely exclusively on penicillin because the former diffuses into the theca and serous cavities and may thus forestall the spread of the infection to these situations a property not possessed by penicillin. The liquefaction and expulsion of secretion from the bronchioles is promoted by the administration of ipecacuanha and similar expectorants. For a child of one year some such combination as tincture of ipecacuanha m 5 and syrup of squill m 3 and camphorated tincture of opium m 3 in a suitable vehicle such as sweetened dill water may be given four hourly in a little hot water.

Cyanosis caused by excessive secretion in the bronchial tract may be relieved by the emetic action of tincture of ipecacuanha m 30, but this treatment is not advisable for weakly patients for these, venesection with the withdrawal of 1 to 2 ounces of blood from one of the antecubital veins is to be preferred. Bronchial spasm is relieved by adding tincture of belladonna m 5 to the above mixture or by giving ephedrine gr $\frac{1}{2}$ once or twice daily. An additional treatment of value for cyanosis and dyspnoea is the use of an oxygen tent, or oxygen may be administered by nasal catheter, or in the case of small infants by a rubber funnel *closely applied*. Inhalation of 7 per cent carbon dioxide in oxygen is of value in impending respiratory failure. High pyrexia is controlled by tepid sponging, which will also promote sleep. Chloral hydrate 1 gr dissolved in a teaspoonful of simple syrup, for a child of one year is the most suitable hypnotic. With the exception of camphorated tincture of opium given as above, no opium preparation is permissible. Circulatory collapse is treated by brandy, 20 drops two-hourly for a child of one year or by a hot mustard bath (one ounce of mustard to each gallon of water at 100°F).

Pertussis

The severe bouts of coughing sometimes leading to an asphyxial convulsion may give rise to much anxiety. Relief may be obtained by full doses of tincture of belladonna (up to m 30 daily for a child of one year) phenobarbitone gr $\frac{1}{2}$ twice daily or phenazone gr 1 incorporated in a mixture four hourly. Ether m 30 dissolved in half an ounce of olive oil and introduced into the rectum twice daily has proved of value (*see also page 273*)

Stridor

Alarming dyspnoea and cyanosis with stridulous inspiration and recession of ribs may occur in acute catarrhal laryngitis laryngismus stridulus laryngeal diphtheria acute oedema of the larynx and after the inhalation of a foreign body. An acute retropharyngeal abscess may cause similar symptoms.

Laryngismus stridulus is a manifestation of tetany and facial irritability carpo pedal spasm and evidence of rickets may also be found. pyrexia and catarrhal symptoms are not usually present. Rapid improvement is obtained by giving calcium gluconate 5 c cm of a 10 per cent solution intramuscularly. The treatment of rickets should be started without delay.

Diphtheria, affecting the larynx is an immediate threat to life. A toxic appearance offensive breath enlargement of the cervical lymph nodes and sanguineous nasal discharge are suggestive accompaniments. In all cases of stridor the throat should be carefully inspected for membrane and a swab taken if the least suspicion of diphtheria exists. There should be no delay in the treatment if diphtheria is a possibility on clinical grounds and at least 32 000 units of anti diphtheritic serum should be given at once preferably by the intravenous route the dose may need to be repeated after 12 hours. If cyanosis increases preparations must be made for tracheotomy.

Inhalation of a foreign body may call for urgent measures if it is impacted in the larynx (*see page 90*). The help of a laryngologist may be needed for its removal through a bronchoscope.

Inhalation of steam from the spout of a kettle has on occasion led to acute oedema of the larynx necessitating tracheotomy.

An acute retropharyngeal abscess is caused by pus tracking behind the posterior wall of the pharynx it forms a boggy swelling which causes dyspnoea of a choking character. The child adopts a characteristic attitude sitting with neck extended the chin supported on the hands and the elbows on the table. The diagnosis is made by palpation through the mouth and treatment consists in incision of the posterior pharyngeal wall the child being in the supine position with the neck hyperextended over the end of the table. Gravity can then assist the cough reflex to prevent aspiration of pus.

(Chronic retropharyngeal abscesses also occur they are usually of tuberculous origin but do not cause urgent symptoms and should not be incised.)

CARDIO VASCULAR SYSTEM

Acute cardiac failure

Pallor vomiting orthopnoea tachycardia and an anxious expression are danger signals in a child suffering from the toxic myocarditis of acute rheumatism. The first sound at the mitral area is softened and an apical systolic murmur may be audible. Cardiac dilatation is indicated by epigastric pulsation and an increase in cardiac dulness. Pulsation may be seen in the engorged jugular veins and the liver enlarges and is tender. Cough and basal crepitations indicate congestion in the pulmonary circuit. Pericarditis is an accompaniment of the severer forms of rheumatic carditis and the soft pericardial friction best heard towards the base of the heart should be listened for daily. Subsequently pericardial effusion may develop causing an increase in the cardiac dulness and depending on the size of the effusion aggravation of all the symptoms and signs already described.

Pericarditis may also occur as a complication of pneumonia osteomyelitis and the septic states associated with bacteraemia. In these conditions the effusion is frequently purulent and its presence may be overlooked on account of profound toxæmia—a serious omission because urgent treatment may well be required.

When rheumatic carditis is of such severity as to cause orthopnoea the supine position normally enforced in milder cases must be abandoned and the child should be propped up on pillows. Glucose 5 per cent in lemonade will suffice for nourish

ment when symptoms are grave but as improvement sets in small feeds of Benger's food bread and butter or plain biscuit with honey or syrup egg custard or milk pudding are allowed four hourly. If œdema is present it is wise to restrict milk because of its relatively high salt content.

Restlessness is a serious symptom calling for speedy treatment. Pain and anxiety are best relieved by opium e.g. powder of ipecacuanha and opium B.P. gr. 1 or solution of morphine hydrochloride B.P. m. 1 for each year of life repeated four to six hourly as required. Kaolin poultice B.P. is applied to the præcordium. Vomiting may be checked by drop doses of weak solution of iodine B.P. and cough by Gee's linctus (Compound Linctus of Squill B.P.C.). Salicylates are of no value except in carditis associated with rheumatic fever when they should be given in doses adequate to control fever and the joint manifestations. Digitalis and other drugs having a similar action are disappointing in the heart failure of acute carditis and may do harm if given to the point of full digitalisation. An attempt may be made to rally a rapidly failing circulation by injecting subcutaneously strychnine hydrochloride gr. $\frac{1}{64}$ nikethamide solution 1 c.cm. or solution of adrenaline hydrochloride 1 in 1 000 0.5 c.cm. A rapidly accumulating pericardial effusion constitutes an immediate threat to life and should be relieved by paracentesis (page 381). The fluid should be examined and cultured and if it is infected 250 000 units of penicillin should be injected into the pericardial cavity.

(For heart failure in acute nephritis see page 228.)

Anæmia

Anæmia in children and infants is liable to be more rapidly progressive than in adults. Lassitude is an early symptom and there may also be jaundice which tends to mask the pallor which would otherwise be readily apparent. Vomiting is of grave import since it may indicate the onset of a hæmolytic crisis. Urobilinuria biluria or hæmoglobinuria may occur according to the severity of the intravascular hæmolysis which has taken place. The child's life is in danger if the red cells have rapidly fallen to 2 000 000 per c.mm. or if the hæmoglobin has dropped to below 30 per cent. Low figures of this nature are likely to be encountered in icterus gravis neonatorum acute and chronic hæmolytic anæmia aplastic anæmia and leukaemia. Hæmorrhage

external or internal may also lead to serious anæmia it may be caused by trauma or by hæmophilia purpura and hæmorrhagic disease of the newborn Exact diagnosis is desirable but in the absence of adequate laboratory facilities there should be no delay in resorting to blood transfusion when this is clearly needed In no condition is accurate hæmatological assessment more necessary than in hæmolytic disease of the newborn (erythroblastosis foetalis) and the appropriate investigations are commonly undertaken by those responsible for the blood bank When jaundice anæmia or oedema are apparent soon after birth and there is a maternal history of stillbirths or of previous infants suffering with neonatal jaundice hydrops foetalis or congenital anæmia erythroblastosis at once suggests itself especially if the liver and spleen are found to be enlarged The disease is now known to be caused by sensitisation of an Rh negative mother by her Rh positive foetus with the result that maternal agglutinins lead to hæmolysis of the newborn infant's red blood cells

Congenital syphilis and neonatal septicæmia may both produce a clinical picture which is somewhat similar to hæmolytic disease of the newborn In the first or second pregnancy the diagnosis is by no means easy but jaundice with increasing pallor should certainly suggest that the necessary blood investigations be carried out To delay transfusion is dangerous and this mistake may be made if cases of so called physiological jaundice are not subjected to critical reconsideration More than one transfusion of Rh negative blood of the appropriate ABO group may be required

In hæmolytic anæmia of other types acute or chronic blood transfusion repeated as necessary will prolong life until remission or spontaneous recovery occurs but in leukæmia its effects are transitory Special care is needed in young children to avoid overloading the circulation by injecting blood too rapidly 10 c cm per pound of body weight represents a safe limit given at the rate of approximately 40 c cm per hour

Hæmorrhagic disease of the newborn, usually manifested by melæna but sometimes by hæmatemesis by umbilical or urinary hæmorrhage is attributable to vitamin K deficiency which leads to insufficient production of prothrombin in the liver Formerly these cases were treated often with success by the intramuscular

injection of 10 c cm of freshly drawn whole blood but it is now rational to make good the deficiency directly by giving the homologue of the vitamin menaphthone 1 mgm intramuscularly (See also page 141) As the response is not immediate blood transfusion may also be needed One ounce of glucose water can be given two hourly by mouth but feeding should otherwise be suspended for 24 hours Hæmorrhage from the vagina without bleeding from any other site (menstruation of the newborn) may be regarded as physiological and does not require treatment

For hæmophilia purpura and agranulocytosis see Chapter IX

NERVOUS SYSTEM

Convulsions

Convulsions frequently accompany acute illness in infancy and early childhood Only in the minority of cases do they indicate intracranial disease and the cause must be sought in such diverse conditions as indigestion acute infections with mounting pyrexia or rickets Disorders such as teething so trivial in themselves as to constitute no threat to life may by provoking repeated convulsions in susceptible children produce a condition of considerable danger as the child may die from asphyxia cardiac failure or hyperpyrexia It is important therefore to treat convulsions symptomatically without delay Chloral hydrate gr 1 up to six months and gr 2 up to 12 months is given by mouth if consciousness is sufficiently recovered between spasms to allow the child to swallow Alternatively sodium phenobarbitone gr $\frac{1}{2}$ may be injected intramuscularly When the fits follow one another in rapid succession an infant should be immersed in a mustard bath which is prepared by the addition of one oz of mustard made into a paste to each gallon of water at 100°F If the convulsions still persist paraldehyde 0.25 c cm is given by intramuscular injection A lumbar puncture should be performed in the more refractory cases and when there is reason to suspect that meningitis may be present A simple enema is administered since relief of constipation in this way may terminate the convulsions It will also prepare the rectum for the absorption of chloral hydrate (in double the oral dose) In long continued convulsions the rectal temperature may rise to 105°F or more and a wet pack may be required to reduce it The same principles of treatment

as relevant apply to status epilepticus in older children. In infants over the age of six months the possibility that the convulsions are associated with rickets and are directly attributable to lowering of the ionised calcium in the blood should not be overlooked. In these cases the fits are a manifestation of tetany and will be promptly relieved by the intramuscular injection of 5 c.c.m. of a 10 per cent solution of calcium gluconate.

Coma

Occasionally babies and older children who have previously seemed in good health may become comatose as a result of a convulsion which has not been observed or when the head has been injured causing concussion or meningeal hæmorrhage. In such cases the person responsible for the care of the child may fail to report the injury or convulsion through fear of censure. Coma may also be caused by fulminating meningitis or encephalitis when retro nuchal rigidity will be a feature. Cerebral hæmorrhage may occur apart from injury from a congenital aneurysm of the cerebral vessels or from the rupture of a vessel in a hitherto symptomless cerebral tumour. Cerebral thrombosis particularly of the superior longitudinal sinus may occur in debilitated infants and may produce convulsions with coma and rapid death. Rapidly fatal coma also occurs in suprarenal hæmorrhage which may result from birth trauma or may supervene in the course of acute infections particularly meningococcal septicaemia (see page 160).

In children who have recently arrived in this country from abroad it is well to remember that coma may be a sign of malignant tertian malaria. In all such cases quinine should be given intramuscularly or intravenously without delay a suitable dose for a child of 5 years would be 4 grains of quinine bihydrochloride repeated if necessary in four hours.

Paralysis

It sometimes happens that a child previously in good health or following a mild febrile disorder loses the use of his limbs. In infants a painful condition of the bones may lead to apparent paralysis before the age of six months this may be the result of syphilitic epiphysitis and after this age scurvy. Palpation or movement of the limbs causes screaming and with the help of a

careful history and by searching for concomitant signs it should not be difficult to establish a diagnosis. After the first year sudden paralysis should suggest **poliomyelitis**. In this condition signs of slight meningeal irritation are present at the onset and there is flaccid paralysis with no sensory involvement. Another common cause of paralysis at the same age period is **diphtheritic polyneuritis**. Enquiry will often elicit the history of a sore throat a few weeks previously the significance of which may not have been fully realised. Some anæsthesia of the hands and feet is usually present but the most typical sign is palatal paresis with nasal speech and regurgitation of fluids through the nose. Signs of toxic myocarditis may appear and the only safe course to adopt is to insist on strict recumbency until the improvement is obvious. Anti diphtheritic serum may be given but at this stage it is practically valueless. 20 000 units intramuscularly would be a suitable dose.

Other forms of peripheral neuritis are rarely seen. **Infective polyneuritis** may lead to an ascending paralysis of the Landry type with involvement of the respiratory muscles. In all forms of paralysis mentioned above it is well to know where a mechanical respirator can be obtained if need should arise (*page 407*).

It remains to mention **paralytic chorea**. In this rare form of a common disease there may be suspension of all voluntary movements but respiration is not affected and there is no danger to life. Diagnosis can usually be made by observing slight choreiform movements in certain muscle groups.

Two other urgent conditions prompt treatment of which may obviate surgical intervention must also be mentioned namely **paraphimosis** and **acute otitis media**.

PARAPHIMOSIS

The earlier an attempt is made to draw forwards the œdematous foreskin the more likely it is to be successful. The distal portion of the penis should be wrapped in a compress of cotton wool soaked in 0.1 per cent solution of adrenaline hydrochloride for 10 minutes. The penis is then held as shown in Fig. 18 and the prepuce restored to its normal position. If this manœuvre fails an operation to release the constricting band will be necessary.

ACUTE OTITIS MEDIA

Earache caused by acute otitis media complicating an upper respiratory infection in a child is a frequent cause of parental anxiety and an urgent call for the doctor

Examination with an electric auriscope reveals an inflamed eardrum—pink in the early stages and red and bulging later. The choice to be made is whether to use chemotherapy or not. Sometimes the decision to do so is easy for there are severe general

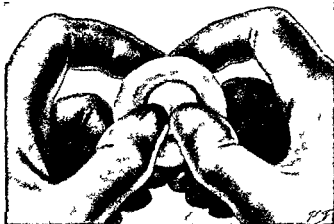


FIG. 18

Method of attempting to reduce a paraphimosis
(*See Surgical Handcraft*)

symptoms or even signs of meningitis. Generally speaking chemotherapy should be used in all cases except those seen late with established discharge and in whom mastoiditis is probable.

The sulphonamide of choice is sulphamezathine and it should be given in full dosage (*see page 440*). Alternatively penicillin may be used (*see page 442*). Within 24 hours the pain is relieved but treatment should be continued until the temperature has been normal for 48 hours though not longer than 6 days in all.

The danger of chemotherapy is that it may mask symptoms. Although earache disappears the drum must be inspected since if it remains opaque and bulging myringotomy may still be required.

Similarly a purulent discharge and deafness may be the only evidence of mastoiditis the other signs being masked.

Myringotomy is indicated if the drum remains opaque and

bulging Under general anæsthesia with nitrous oxide or thiopen tone the meatus is cleaned with 70 per cent spirit and the largest speculum which gives a full view of the drum inserted A myringo tome (Fig 19) is then introduced down the speculum and the

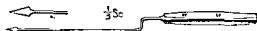


FIG 19
Myringotome



FIG 20
Showing site of incision
through the right tym-
panic membrane
(After Surgical Handcraft)

drum incised from below upwards in its posterior part (Fig 20) Pus and blood are mopped away An anæsthetic may be omitted particularly if the drum is bulging

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R W BROOKFIELD

CHAPTER XVII

Emergencies in Infectious Fevers

General considerations

IN fever practice problems demanding immediate decision and action arise daily as every patient admitted is at some period potentially infectious. Naturally such problems may occur in any medical practice private or public but in fever hospitals almost every patient presents a problem which may be impossible of immediate solution if individual isolation accommodation is limited and the numbers seeking admission high. Our control of epidemics is still imperfect, an error of judgment or lack of due care may have serious consequences for the particular patient the immediate contacts or the community.

In the field of therapeutics prompt measures for combatting toxæmia as seen in meningococcal invasions or for relief of urgent symptoms in laryngeal diphtheria or acute poliomyelitis are required to avert death or prevent permanent disability. Not infrequently, some emergencies are found to be surgical and not strictly medical—a matter of erroneous diagnosis and notification. Acute appendicitis may be mistaken for enteric fever intussusception for enteritis or dysentery osteomyelitis or suppurative arthritis for scarlatinal rheumatism and ruptured ectopic gestation for puerperal peritonitis.

It must not be assumed that these errors are necessarily diagnostic blunders on the part of the practitioner. They are rather abuses of notification used to facilitate admission to hospital.

Emergency measures pending removal to hospital include the use of sedatives, specific sera, sulphonamides and penicillin the dose and time of administration of which should always be recorded.

Most medical emergencies in fever practice fall into two main groups (1) acute toxæmia (2) urgent dyspnoea. The same patient may show features common to both—as in laryngeal diphtheria suffocative pneumonia of measles and whooping cough and anuric coma of scarlatinal nephritis. Acute toxæmia and

dyspnoea will be briefly considered first and then the special emergency aspects of certain fevers

ACUTE TOXAEMIA

This may be present from the onset or develop gradually. Cerebration may appear normal or all gradations from slight disorientation to established toxic psychoses may appear both in the initial stages or as a terminal event. Drowsiness and stupor pass into coma usually accompanied by obvious respiratory arrhythmia and distress. Dehydration and inanition may be early and severe especially in infants and the aged or gradual in development with subsequent sudden exacerbation.

DIAGNOSIS—The best opportunities for successful therapy have often passed before accurate diagnosis is possible. Hence to avoid mistakes the diagnostic net must be large as well as fine.

Investigation of the metabolic disorder consequent upon invasion as well as the nature of the infecting agent is carried out more or less on the same lines as in any other clinical condition. The thermometer is useful in excluding non febrile disorders but may be misleading since normal or subnormal temperatures may accompany the toxæmia of infective disease. Acute septicæmic or meningitic conditions may be closely imitated by uræmia, diabetic coma and cerebral vascular lesions to mention a few common instances. The presence of a cutaneous rash usually of diagnostic help may mislead as most eruptive fevers exhibit considerable variation both in the general pattern of the rash and the appearance of individual lesions. This applies especially to prodromal rashes notably the bathing pants hæmorrhagic rash of smallpox and the urticarial and scarlatinai prodromes of measles. Early diagnosis is important also in instituting appropriate administrative measures for the control of spread.

In obscure conditions which cannot be diagnosed outright it is often found that the optimum time for taking particular specimens or applying specific tests has already passed before the results of the preliminary findings have suggested the true nature of the disease. Foresight is therefore necessary in collecting specimens. Sufficient blood for example should be taken (say 10 c cm) to allow of whatever tests are needed at the time or

are likely to be needed later. Half should be allowed to clot (for serum) and the remainder oxalated (for plasma).

Treatment—Treatment of toxæmia may be summed up by saying that it must restore the disordered metabolism to normal as rapidly and completely as possible. In a general way the human body responds to all toxæmias and infections in more or less the same manner. Treatment must therefore be largely restorative supplemented by such prophylactic and therapeutic agents as may be available commonly, immune sera (antitoxic or antibacterial) and/or chemotherapeutic drugs singly or in combination. *Glucose hydrolysed proteins and vitamins* may be given orally to supplement the ordinary diet or in special cases intravenously.

SEROTHERAPY—As far as is known *antitoxic* sera do not influence the action of specific drugs favourably or otherwise except in so far as they neutralise circulating toxins and thereby aid the patient to overcome the infection. On the other hand *antibacterial* sera may interfere with the action of penicillin (but not sulphonamides) by inducing a dormant (*persistor*) state in which organisms may become temporarily insusceptible to the drug. Clinicians have generally abandoned the use of the antibacterial sera in the belief that they are now unnecessary and potentially dangerous.

CHEMOTHERAPY—The dosage and methods of administration of sulphonamides, penicillin and streptomycin are described under Practical Procedures (Chapter XXIII).

URGENT DYSPNŒA

When this complicates infectious fevers it is caused by —

- (1) Mechanical obstruction from spasm, œdema and exudation (*usually diphtheritic* pages 258 and 270)
- (2) Paralysis of respiratory muscles as in diphtheritic neuritis and poliomyelitis
- (3) Pulmonary disease (*e.g.* pneumonia)

Paralysis of the respiratory muscles may call for the use of a breathing machine, details of the use of which are given on page 407. Dysphagia is a frequent accompaniment and the operator may be tempted to incline the machine to allow secretions to drain away. This may be done safely for short periods but prolonged tilting leads to œdema of the brain. Hence it is

safer to avoid tilting and withdraw secretions by a catheter attached to a suction pump. Because of the risk of pneumonia it is wise to start prophylactic chemotherapy.

Urgent dyspnoea resulting from bronchitis and pneumonia may complicate many infectious fevers, particularly measles and whooping cough in the very young. There is usually considerable toxæmia also. Measures to be adopted are described elsewhere (*oxygen therapy* page 409 *penicillin by injection and inhalation* page 442 *sulphonamides* page 441).

SPECIAL URGENT FEATURES OF CERTAIN FEVERS

Scarlet fever.

At the present time scarlet fever is so mild as rarely to constitute an emergency except when mastoiditis or anuric nephritis suddenly changes the clinical picture. These complications should be exceedingly rare providing antitoxic serum (3 000 to 6 000 U.S.A. units contained in 3 to 6 c.cm. for a child of five years) is administered at the outset together with sulphonamides [sulphanilamide being the most commonly used in moderate doses (*page 440*) for five days]. In toxic or septic attacks *serum dosage should be doubled and preferably given intravenously* (after performing intradermal sensitivity tests and desensitising if necessary, *page 27*), together with penicillin and sulphonamides. A special advantage of penicillin therapy is that elimination of the causative organisms from the upper respiratory tract is rapidly achieved usually in four to five days. This allows early release from isolation: the period nowadays is as short as 14 days—in the absence of complications. For the treatment of nephritis with anuria *see page 230*.

Sore throat.

When a doctor is called to a patient complaining of a 'sore throat' two serious conditions for which treatment must be instituted immediately should be at the back of his mind. They are diphtheria and—a rarer condition—agranulocytosis (*page 139*).

Diphtheria.

The nation-wide immunisation campaign pressed vigorously during the war years has yielded a handsome dividend in the virtual disappearance of clinical diphtheria but while less than

75 per cent of the population at risk are immunised severe cases may occur. Mild tonsillar and nasal cases require 5 000 to 10 000 units of antitoxin intramuscularly moderate invasions 10 000 to 25 000 units and late malignant attacks will require 100 000 units or more at least half of which should be given intravenously. Whilst adults may need larger amounts it should rarely be necessary to repeat the dose especially since penicillin has become available as an adjuvant. Whilst it must be confessed that a clear cut therapeutic effect is rarely encountered with penicillin it should always be used in toxic cases. Nourishing food is essential but there does not appear to be any advantage in a high carbohydrate intake with or without insulin or in intensive vitamin therapy unless a deficiency existed before the attack. Adequate fluids by mouth or nasal catheter or parenterally if vomiting dysphagia or coma complicate the picture using saline glucose and plasma are essential in all severe cases. The amounts and concentrations depend on the need as revealed by the presence of œdema and fall in plasma protein level (normal range 6.5 to 8.5 gm per cent). Any good effect of nikethamide given four to six hourly in doses of 0.25 to 0.5 c cm for a child of five years is temporary as is that of adrenaline or ephedrine. Digitalis is inadvisable on account of the frequency of heart block of varying degree in diphtheritic myocarditis. The cold clamminess and pallor of the limbs from peripheral vasoconstriction is best remedied by heat by hot packs or by immersing the patient in a hot bath. The effect is usually only temporary. Restlessness insomnia and distressing precordial pain may require morphine for complete relief.

Acute circulatory collapse in diphtheria commonly results from heart block (page 271) or from a fall of blood pressure caused by hæmorrhage into the suprarenals (Waterhouse Friderichsen syndrome page 225).

Laryngeal diphtheria

This dreaded *morbus suffocans* of older writers was until recently the commonest cause of respiratory obstruction threatening life. The diagnosis from the more frequent coccidial laryngitis complicating measles or the common cold can only be made with certainty by direct laryngoscopy. The technique

of the examination suction of false membrane and intubation can best be learnt in a properly equipped centre such as is available nowadays in most large fever hospitals.

Sedatives such as phenobarbitone and drugs to relax laryngeal spasm such as ephedrine may be used but only under continuous skilled supervision. If in doubt one should operate early. It is probably still true that tracheotomy is more frequently practised than intubation in Great Britain.

EMERGENCY TRACHEOTOMY—For the practitioner without special equipment tracheotomy is usually the only means at his disposal in an emergency: a sharp scalpel, an artery forceps to act as tracheal dilator and a catheter or stout rubber tubing of suitable size are useful substitutes for the conventional instruments. Provided the larynx is held firmly between the left thumb and midfinger, the forefinger being placed on the cricoid and the incision is made downwards exactly in the middle line, little difficulty need be anticipated even although the operator has never witnessed the operation before—as frequently applies to the recently qualified doctor. No time should be wasted in securing hæmostasis: free bleeding is a good sign and affords considerable relief. As the patient after tracheotomy needs highly skilled nursing, he should be transferred to a suitable hospital as soon as possible, the doctor preferably accompanying him in the ambulance.

Measles

The prodromal or pre-eruptive stage of measles may be accompanied by alarming symptoms of laryngeal obstruction (croup) and by considerable toxæmia; indeed it is usual for the patient to feel better as the rash appears. While some patients are prone to develop laryngitis with any respiratory infection (even the common cold), the onset of croup should suggest a secondary invader. This is usually the hæmolytic streptococcus but occasionally the diphtheria bacillus. Therefore diphtheria antitoxin should be given in all but the mildest grades and in severe attacks sulphonamides and/or penicillin also. The latter will not only help to arrest laryngeal inflammation but may prevent the onset of broncho-pneumonia. Steam kettles or tents and poultices and even oxygen therapy are of secondary importance. Phenobarbitone in doses of $\frac{1}{2}$ gr preferably with

5 gr sodium bromide four hourly for a child of five years may avert the need for tracheotomy but morphine is contra indicated generally Ephedrine $\frac{1}{2}$ gr to ease spasm with or without atropine $\frac{1}{150}$ gr to dry up secretions may be tried Caution is needed in using atropine lest tenacious plugs of muco-pus result which weakly subjects may be unable to dislodge

Measles encephalo-myelitis

Although improved nutrition and hygiene of the susceptible population and timely chemotherapy have resulted in a striking fall in the complication and fatality rates of measles in recent years a notable exception is acute disseminated meningo-encephalo myelitis which seems to be as common though perhaps not as fatal as ever (The true incidence is unknown as unlike measles itself it is not notifiable)

In general the severity of this complication is related to the severity of the primary disease The incidence is about 1 in 2 000 cases and the case mortality about 10 per cent The onset is marked by headache vomiting and rapidly increasing drowsiness 7 to 14 days after measles begins Cerebral and spinal forms have been described Diagnosis is confirmed by lumbar puncture which reveals a fluid usually under slightly increased pressure containing excess protein and mononuclear cells (20 to 100 per c mm) Glucose and chlorides are not reduced thus differentiating the condition from tuberculous meningitis

Treatment—Lumbar puncture intravenous hypertonic (20 to 25 per cent) dextrose and intramuscular 10 per cent calcium gluconate in doses according to age and severity are the usual measures employed For an infant 5 per cent calcium gluconate should be used as the higher concentration may cause muscle necrosis If the disease progresses convalescent measles encephalitis serum if available from a recovered patient should be given intravenously in doses of 10 to 50 c cm according to age Failing this gamma globulin (Lister Institute) or convalescent measles serum (not generally available but many hospitals hold small stocks) may be tried

Whooping cough (See also page 258)

Whooping cough has replaced measles as the worst enemy of childhood mainly by reason of the severe irreparable damage

inflicted on the lungs even by a mild attack. Broncho pneumonia complicating whooping cough is a medical emergency and calls for prompt treatment by sulphonamides and/or penicillin.

In infants especially convulsions (*page 262*) and gastro-enteritis (*page 250*) are urgent complications and constitute specially dangerous threats to life. Breast feeding phenobarbitone and sulphonamides are the chief measures both in prevention and treatment of these complications.

Cerebrospinal fever

Sudden onset of coma especially with a purpuric rash should suggest spotted fever even before the onset of meningitis. Blood should be taken for culture putting 5 c cm of blood into 50 c cm of serum broth and prompt intensive sulphonamide therapy (sulphathiazole or sulphadiazine) started. There is no evidence that penicillin gives better results. With modern treatment the case mortality is under 10 per cent being very low (1 to 2 per cent) in young adults but high in infants and those over 50 years. Nevertheless fatal fulminating attacks with extensive purpura adrenal hæmorrhages and very occasionally encephalitis may occur at any age. Occasionally fluid blocks occur at the foramen magnum the iter or over the cortex necessitating the special measures which are more frequently needed in other pyogenic forms of meningitis.

Pneumococcal meningitis

Intensive chemotherapy using sulphadiazine or sulphamerazine is required from the onset. In addition penicillin should be given systemically and intrathecally. If blockage occurs the spinal canal should be washed out with saline through needles inserted at the cisternal and lumbar sites. Introduction of highly purified penicillin in high dilution (1 000 to 3 000 units per c cm) into the ventricles and the subarachnoid space or over the hemispheres may be required in desperate cases. Not more than 10 c cm should be inserted at a given site daily or on alternate days until recovery. The help of a neurosurgeon should be sought.

Influenzal (Pfeiffer) meningitis (*See also page 162*)

This serious disease fortunately infrequent in this country but common and on the increase in America is an emergency calling for intensive therapy. Although successes have been recorded with specific Pittman B anti serum prepared from rabbits

combined with sulphonamides (preferably sulphadiazine), and later with serum and penicillin, the mortality remains high. The results with streptomycin have been somewhat disappointing. Anti-serum is obtainable through Messrs Savory & Moore (143 New Bond St, W 1, Tele MAYfair 4471) or through the Public Health Laboratory Service (38 Old Queen St, Westminster, S W 1, Tele WHItchall 4884).

Typhoid fever.

A combination of penicillin and sulphathiazole is recommended. A course of 10 mega units of penicillin and 34 grams of sulphathiazole in four days should be given and repeated after a two-day interval. Alternatively, Felix's vi anti serum (Allen and Hanbury, 7 Vere St, W 1, Tele MAYfair 2216, or the Lister Institute, Elstree Herts, Tele Elstree 1009) should be used, giving 30 to 50 c cm intramuscularly on three to five successive days.

Hæmorrhage and perforation are relatively rare complications in Great Britain nowadays but may still be met with abroad.

Hæmorrhage, which occurs in 5 to 10 per cent of cases, varies from a mere oozing to the sudden loss of several pints manifested by pallor, rapid thready pulse, sighing respirations and a fall of temperature. There may be pain or a sensation of something giving way in the abdomen. If the patient is constipated, blood may be passed only after a long interval. Treatment is on the usual lines for shock, by morphine and blood transfusion.

Perforation is nowadays a rare complication of typhoid because the improved dietetic regimen has almost eliminated severe meteorism. It occasionally complicates an apparently satisfactory case. The usual onset is with sudden pain, tenderness and rigidity in the right iliac fossa, but these features may be masked in the presence of severe toxæmia and semi-stupor. After a brief fall in temperature and apparent improvement following the initial shock of perforation the temperature rises with the onset of generalised peritonitis. Increasing distension, rapid respiration and the Hippocratic facies appear, and unless a successful operation is quickly undertaken death follows in 36 to 48 hours.

Dysentery—Salmonella infections.

(Symptomatic treatment is described on page 252.) When

acute diarrhoea is shown to be caused by one of the dysentery salmonella organisms phthalyl sulphathiazole should be given for about five days. The dose for an adult is 4 gm followed by 2 gm four hourly when diarrhoea is severe and 3 gm followed by 2 gm six hourly when diarrhoea is mild. It appears to be non toxic and the effective dose is smaller than that of sulphaguanidine or sulphasuxidine.

Smallpox

During the war years and subsequently smallpox has been introduced on numerous occasions into Great Britain but only once in 1942 in Scotland did it establish itself sufficiently to require mass vaccination. The short journey by air from endemic areas and the occasional tendency for oriental smallpox (*variola major*) to break through the protection afforded by vaccination as practised at present combine to increase greatly the risk of importation. The *highly modified form in which only some six to twelve lesions are present* may be disregarded by patients and unrecognised by doctors. It can however cause malignant attacks in the unvaccinated the source of which may not be readily traced. Severe influenzal symptoms followed by a few papules which may commence on the trunk and may not pustulate should be regarded with suspicion and the nearest Public Health Laboratory consulted with a view to isolating the elementary bodies and performing the complement fixation test. In the treatment of malignant attacks characterised by prostration and vomiting intravenous glucose saline and plasma should be employed the latter preferably from recently vaccinated subjects together with sulphathiazole and penicillin to combat the effects of staphylococcal pustulation.

EMERGENCY ASPECTS OF PROPHYLAXIS

The need for urgent protection against infection may in the young and debilitated constitute an emergency. By adopting a well planned immunisation programme the number of such emergencies should be greatly reduced.

Smallpox

Immediate contacts should be vaccinated. They may be allowed to travel to their homes but the local Medical Officer

of Health should be informed. Mass vaccination is only called for if the disease is of the major variety and has got a good hold.

Diphtheria

The emergency prophylactic measures to be adopted depend on the circumstances. If there is only one case no action need be taken beyond careful watching of contacts for a week. If further cases arise they should be isolated and contacts Schick tested. If positive they should receive passive or combined passive and active immunisation. Indiscriminate swabbing of contacts is nowadays discouraged and is unnecessary if the herd immunity is high from antecedent immunisation. School closure and exclusion of contacts is rarely practised, being superseded by immunisation and close supervision for early signs of the disease. Occasionally Schick negative subjects may develop diphtheria but the disease is then nearly always mild.

IMMEDIATE PROTECTION BY PASSIVE IMMUNISATION

This may be called for as an emergency measure because the exposed person is already ill or debilitated. Passive immunity is short lived, providing 10 to 14 days complete protection and alleviation of the disease if contracted in the subsequent week or two. It is available for the following diseases —

Diphtheria Give 2 000 to 5 000 units of anti diphtheric serum intramuscularly according to age. It may be combined with active immunisation by giving 0.5 c.c.m. alum precipitated toxoid followed four weeks later by 1.0 c.c.m.

Measles Give convalescent serum 0.2 to 0.3 c.c.m. per lb body weight in the first five days after exposure. As this may not be readily available an alternative is to give parents' blood (0.5 to 1.0 c.c.m. per lb body weight) provided they have had measles. Smaller and later doses will attenuate the attack but as an emergency measure complete prevention is usually desired.

Convalescent serum is also available against rubella, chicken pox, mumps, whooping cough and poliomyelitis, but only against measles is it uniformly reliable. The protective titre of convalescent pertussis serum may be enhanced by previously inoculating with vaccine donors who have had the disease.

(hyperimmune serum) alternatively the serum of immunised rabbits may be employed

PASSIVE IMMUNISATION OF PREGNANT WOMEN

An impressive body of evidence not yet amounting to final proof has been collected to the effect that certain virus infections (rubella measles mumps and influenza) in the mother during the early months of pregnancy may and in the case of rubella usually does damage the foetus. Cataract deafness congenital heart disease and other abnormalities may result.

In the first three months the risk is so great that if a pregnant woman contracts rubella therapeutic abortion should be considered. Most large fever hospitals hold stocks of dried convalescent sera against most of these diseases. The average dose is 20 c.c.m. preferably given in the first three days after exposure if given later the dose should be proportionately increased. On account of the possible risk of homologous serum jaundice which cannot be entirely foreseen or avoided human serum especially when taken from a large pool should not be administered indiscriminately or unnecessarily.

ISOLATION AND QUARANTINE

The recognised rules of quarantine have been considerably relaxed in recent years and no useful purpose can be served by trying to formulate rules and regulations to meet all contingencies.

Up-to-date recommendations regarding isolation and quarantine of patients in hospital and their discharge have recently been considered in detail by Mitman who stressed that particular circumstances must govern the appropriate action whether it affects the individual or the group. Legally contacts and carriers cannot be controlled or their liberties restricted apart from prohibition of the handling preparation and cooking of foodstuffs by carriers of intestinal pathogens.

Generally speaking the patient is not infectious during the incubation period and no restrictions on movements are commonly applied. (Infectivity has been proved in the last 24 hours incubation in chicken pox and in the last 2 to 3 days in enteric fever. It may conceivably occur in other infectious diseases.) The quarantine period rarely applied in strict form nowadays is the maximum

incubation period with one or two days added in case the initial phase of the attack is overlooked. Some measure of supervision during the whole incubation period is advisable as contacts may have been exposed to an undetected case or carrier, which gave rise to the first recognised (so called primary) case, and so contract the disease early in the supposed incubation period. Smallpox is the classical example of the official policy of surveillance as opposed to strict imposition of isolation in quarantine and is almost invariably successful when combined with prompt vaccination. For common infectious diseases such as measles, scarlet fever, whooping cough, and diphtheria, similar prophylactic measures are available supplemented by search for the causative organism when practicable and isolation of proved carriers. Mumps and german measles may give rise to considerable trouble especially in schools, barracks and similar semi-closed communities for adolescents and young adults not because of the severity of the disease but because the prolonged incubation periods and relatively low infectivity may lead to persistence for months and interfere seriously with essential activities. Sufficient immune serum is rarely available to protect all immediate contacts. Infantile paralysis (*see also page 172*) may present a serious problem in similar communities, with even more tragic consequences. Again the appropriate action depends on the circumstances and what is best for the individual may not necessarily lie in the interests of the population at large. Break up of a school is the shortest way to abort an outbreak and may be attended by little risk if susceptibles (generally under 25 years) are temporarily removed from the respective homes to which the contacts are dispersed. On the other hand, if the contacts belong to the poorer classes with large young families this measure might prove highly dangerous in converting a single focus into multiple potential foci. Recent experience in Australia has shown the "stay put" measure to be the most effective *method of preventing epidemic spread whether it affects families, isolated communities or large towns*.

EMERGENCY ASPECTS OF DISINFECTION

The term disinfection implies destruction of the *materies morbi* and not sterilisation. In recent years current disinfection

from day to day and virtually from minute to minute has largely replaced so called terminal disinfection at the end of the illness. If the former has been efficient the latter becomes largely superfluous.

Current disinfection

The measures employed naturally depend on the resources available. All contaminated discharges and excreta should preferably be treated for one hour with 2 per cent cetavlon or 5 per cent lysol solution in an amount equal to that of the material to be disinfected. Instruments, crockery and bed pans should be boiled for 10 minutes and fabrics treated with current steam at 230°F for 15 minutes. Some instruments especially if electrical are more conveniently disinfected by exposure to ultra violet light at one foot for one minute.

Terminal disinfection

The end of the illness is a suitable time for disinfecting mattresses, blankets and room furnishings. No special measures are needed for walls and furniture beyond washing with soap and water. Viruses rapidly die after leaving the body but bacteria *e.g.* streptococci and pneumococci may survive for months in dust especially if protected from sunlight.

Transport of infectious cases

Ambulances are sprayed with antiseptic solution (carbolic acid, formalin or hypochlorite). Local authorities have arrangements for disinfecting taxis and other public vehicles should they be contaminated. Bodies of those dying from infectious diseases should be taken direct to the burial place.

Burial

It is not considered necessary to disinfect hearses. It is however advisable that undertakers who have to handle corpses dead from smallpox should be vaccinated or re vaccinated.

WILLIAM GUNN

CHAPTER XVIII

Emergencies in Tropical Medicine

General considerations

SUDDEN illness in tropical practice always demands an intensive and urgent search for its cause since although tropical illnesses may be rapidly fatal if unchecked many of them yield readily to specific treatment early and adequately administered.

The physician should however examine the patient with no special bias towards a tropical infection lest one of the more cosmopolitan diseases be overlooked. This is very important when dealing with natives in whom diseases such as pneumonia and smallpox may be more dangerous than the specifically tropical illnesses to many of which they have some immunity. The finding of a few malaria parasites for example in the blood of a native should not be too readily accepted as the whole explanation of an obviously serious infection. They may be merely incidental and while it is wise to treat the malaria the search should be continued for a more adequate cause.

A complete physical examination is of the greatest importance since the history and even the symptoms described by patients of the primitive races may be vague and misleading. Albuminuria for example might lead to the finding of pus cells and coliform organisms in the urinary deposit and suggest that what presented as malaria was really acute pyelitis perhaps with a kidney tumour simulating a spleen. In all urgent illnesses in the tropics the routine diagnostic procedure should include an examination of the urine thick and thin blood films and direct microscopy of the stool.

When symptoms point to the alimentary canal or abdomen it is well to consider a parasitic disease since some apparently surgical emergencies may have a parasitic basis and may be resolved by medical treatment as for example amœbic typhlitis simulating appendicitis.

Some specifically tropical diseases which may present as emergencies are —

- Pernicious malaria
- Blackwater fever
- Amœbiasis
- Cholera
- Helminthiasis
- Fulminating cardiac beri beri
- Rabies
- Snake bite and other bites and stings
- The effects of heat

PERNICIOUS MALARIA

(For malaria on board ship see page 329)

No emergencies in tropical medicine are more important than those of malignant tertian malaria known as pernicious attacks. Unless recognised early they are fatal whereas if promptly treated therapeutic triumphs result. There are three main clinical varieties.

(a) **Bilious remittent fever** — This is the commonest and least grave form. Characteristic signs are nausea and persistent vomiting, icterus with tenderness and enlargement of the liver, splenomegaly, anæmia and parasitæmia, albuminuria and fever. The persistent vomiting interferes with the absorption of anti-malarial drugs by mouth and unless this is recognised and countered the infection may end fatally.

(b) **Cerebral malaria** — This may develop gradually or suddenly during the course of a malignant tertian infection. In the insidious form there is increasing headache with or without mild delirium, the patient becoming drowsy and finally comatose. At other times coma may be preceded by wild delirium or acute psychotic manifestations while sometimes cerebral malaria is signalled by rapidly progressive hyperpyrexia. Various focal signs may also be produced by blocking of the capillaries of the brain with parasitised cells. (Fig 21)

(c) **Algid malaria** — Here there is profound prostration, a cold clammy skin despite the high internal temperature and frequently diarrhœa and severe anæmia. Cardio-vascular failure is the usual mode of death.

Other clinical forms of pernicious malaria—gastric dysenteric choleraic—are self explanatory They are caused by interference



FIG. 21

Micro-photograph of brain section from a fatal case of malignant tertian malaria. The capillaries are distended and obstructed by parasitized red cells. The heavy black dots in the vessels represent malarial pigment.
x 400

with the local vascular supply by masses of parasitized cells

DIAGNOSIS.—In malarious countries the exclusion of pernicious

malaria is the first step in the differential diagnosis of nearly all acute illnesses

Once the history and symptoms have suggested malaria a competent examination of the peripheral blood should be made to reveal the parasite. In the absence of adequate facilities however parenteral treatment should never be delayed if the possibility of pernicious malaria exists but a few thick blood films should be taken for subsequent confirmation

Treatment—Quinine dihydrochloride 0.5 gm ($7\frac{1}{2}$ grains) should be given intravenously. It is commonly supplied in ampoules in one or two c cm of sterile water and may be so used in an emergency if given extremely slowly. It is better however to dilute it considerably with sterile normal saline. The stronger the solution the slower should be the injection. Two or three injections at six hourly intervals should suffice to control the infection but if anoxæmia of vital centres has been prolonged death may nevertheless occur. It is for this reason that prompt recognition and treatment of pernicious malaria is so essential. The undiluted quinine solution or alternatively mepacrine methane sulphonate 0.3 gm in 5 c cm of sterile water may be given intramuscularly.

Hyperpyrexia should be controlled by cold sponging. Lowering of intracranial tension by lumbar puncture may help comatose patients. Dehydration may call for intravenous glucose saline.

When the acute symptoms have been controlled further treatment should follow—either paludrine 0.1 gram three times a day by mouth for 10 days or mepacrine hydrochloride 0.1 gram three times a day for five days followed by 0.1 gm daily for three weeks.

BLACKWATER FEVER

This dramatic and dangerous complication of malaria affects patients who have long and repeatedly suffered from malignant tertian infections. The attack seems to be precipitated by the administration of an anti-malarial drug—most often quinine.

A sudden intravascular hæmolysis occurs manifested clinically by a chill or rigor, fever, loin pains, vomiting, the passage of dark red or black urine and the early onset of jaundice. The rapid destruction of erythrocytes produces profound anæmia with

the liberation into the plasma of oxyhæmoglobin. A small portion of this is excreted in the urine where it is converted into methæmoglobin. The remainder of the hæmoglobin in the plasma forms methalbumin which is picked up by the reticulo endothelial system.

The urine usually acid contains albumin oxyhæmoglobin methæmoglobin and a considerable deposit consisting of casts renal epithelium and an amorphous chocolate coloured material generally regarded as acid hæmatin. Interference with renal function is shown by blood nitrogen retention and acidosis. Renal failure was formerly thought to be due to blockage of the tubules by precipitated acid hæmatin but though this may be a factor intra renal vascular changes producing cortical anoxæmia probably play an important role. If a sufficient number of nephrons remain functioning the high blood urea produces polyuria but more commonly renal impairment leads to oliguria and even anuria.

The attack may end after a single hæmolytic crisis or repeated waves of hæmolysis may occur. Death may result from acute anæmia or hyperpyrexia or later from renal or circulatory failure. During hæmolysis parasites usually disappear from the blood.

DIAGNOSIS—An acute febrile crisis associated with hæmoglobinuria and jaundice in a patient suffering from malaria suffices in the absence of other known hæmolytic influence to establish a clinical diagnosis of blackwater fever. The presence of hæmoglobin in the urine may be rapidly detected by the spectroscope hæmaturia being excluded by microscopic examination.

Treatment—In the present state of knowledge it is probably wise to suspend anti malarial drugs. If however persistence of parasites forces the issue paludrine or mepacrine may be given. Complete rest is essential all unnecessary transportation being avoided. Steps should be taken to maintain a high fluid intake and to keep the urine alkaline. Sodium citrate or bicarbonate 1 to 2 gm (15 to 30 grains) should be given two or four hourly. Should vomiting interfere with fluid intake or urinary suppression threaten or it be found impossible to alkalinise the urine intra venous infusions may be required. (See *also* page 423.) 500 c cm of normal saline containing 1 per cent sodium bicarbonate may be given every 12 hours. This solution is best sterilised

by filtration as boiling may convert bicarbonate into carbonate which is toxic. It should be added that doubts have recently been raised about the wisdom of excessive alkalisation in renal failure and some physicians prefer normal saline or 5 per cent dextrose for intravenous use. A fluid intake and output chart should be kept and the volume reaction and appearance of each specimen of urine recorded.

In view of the possibility of renal failure being due to renal anoxæmia from dynamic vascular changes bilateral posterior splanchnic block may be of use (see page 385).

If anæmia threatens life blood transfusion should be considered. It may also provide some anti hæmolytic factor but sometimes it seems to contribute to further hæmolysis.

For persistent vomiting reliance should be placed on fluids administered parenterally the multiplicity of other measures (iced champagne drop doses of iodine epigastric stupes etc.) is in itself a measure of their ineffectiveness.

INSTRUCTIONS FOR CONVALESCENCE—Because of the danger of late cardiac failure early exertion should be avoided. The malarial infection may relapse and to forestall this paludrine 0.1 gm. three times a day for 10 days or mepacrine 0.1 gm. three times a day for five days followed by 0.1 gm. daily for three weeks should be given as soon as the hæmolysis ceases.

Provided recovery is satisfactory there is no reason why the patient should leave the tropics but he should avoid further malignant tertian malaria by taking while exposed to the risk of infection suppressive treatment e.g. mepacrine 0.1 gm. once daily or paludrine 0.1 gm. twice weekly or chloroquin 0.2 gm. weekly.

AMCEBIASIS

While this disease does not give rise to emergencies comparable in urgency with those of malaria its complications demand prompt treatment if the patient's health and even life are to be preserved.

Hepatic amœbiasis

This is the most important complication of infection by *Entamoeba histolytica* and may occur with or without intestinal symptoms. The earliest lesion is a diffuse hepatitis which may develop through the stages of microscopic abscess formation the

main incidence being in the right lobe until an abscess occupies the greater part of the viscus. A single abscess dominates the picture although multiple lesions are frequent.

The symptoms and signs may develop insidiously or be detected at any stage of the infection and they depend on the

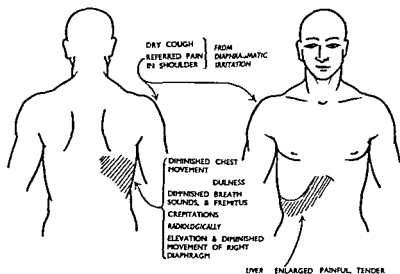


FIG 22

Amoebic infection of liver

nature and age of the lesion. In the early stages there may be only irregular fever with discomfort or tenderness in the region of the liver accompanied by a neutrophil leucocytosis. Later enlargement of the liver occurs with increased pain and tenderness over it. There may also be pain in the shoulder and a dry cough from diaphragmatic irritation. Fever and leucocytosis become more pronounced, chills and sweats develop. The patient loses weight and his complexion becomes sallow and muddy. With progression of the lesion, limitation of respiratory movement of the upper abdomen and lower chest appear. Dulness and rales may be found indicating collapse of lobules in the lower lobe of the lungs (Fig 22). Deformity, elevation and immobility of the diaphragm may be demonstrated radiologically (Fig 23).

If the condition is not recognised and treated, progressive toxic deterioration of the patient's condition occurs. The abscess may rupture into a bronchus with expectoration of blood stained pus

and liver debris—the characteristic anchovy sauce sputum into the peritoneal cavity usually with a fatal result into the intestine or externally (Fig 24) Metastatic lesions may develop in distant organs with grave consequences



FIG. 23

A liver abscess causing deformity and elevation of the right dome of the diaphragm

(D. C. R. n. Inst. Lo. U. J. courtesy of S. I. Manson-Ball)

DIAGNOSIS—This depends largely on the clinical picture of fever, high neutrophil leucocytosis with signs pointing to the liver in a patient who may have been exposed to amœbic infection. The presence of *E. histolytica* in the faeces adds to the evidence, but their absence does not negative the diagnosis. In the later stages diagnostic puncture of the liver will reveal characteristic chocolate coloured pus, but amœbæ may appear in it only after it has been draining for some days as they live in the wall of the abscess cavity. At the stage when the disease should be recognised, namely that of hepatitis or miliary abscess formation, a presumptive diagnosis may be made by the response to emetine.

Treatment—Emetine hydrochloride 65 mgm (1 grain) daily should be given subcutaneously for 10 days. In the earlier stages

of hepatitis it produces a marked improvement in two or three days

If there is abscess formation emetine alone may be insufficient and evacuation of the abscess may be necessary in addition. This should be done by aspiration under local anæsthesia. The site is determined by the area of maximum tenderness swelling œdema

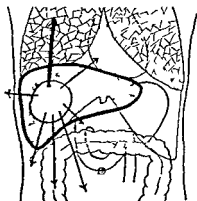


FIG 24
Directions in which a tropical
liver abscess may burst
(After Cope)

or crepitus. If there is no such lead the needle should be inserted in the eighth or ninth interspace in the posterior axillary line since most abscesses are in the upper and posterior part of the right lobe. A Potain's aspirator and a large bore needle (No 9 bore) are generally used as the pus may be very thick. The needle should be inserted in various directions until pus is obtained but not deeper than $3\frac{1}{2}$ inches lest the inferior vena cava or portal vein be punctured. Aspiration should go on until the cavity is emptied. Great care must be taken to avoid secondary infection of the cavity. Should this occur penicillin may be indicated.

TOXIC EFFECTS OF EMETINE—The patient will of course be confined to bed but in view of the possible effects of emetine on the myocardium he should also be prohibited from unnecessary exertion. The toxic effects of therapeutic doses appear to have been over emphasised but should be considered if there is a rising pulse rate or unduly falling blood pressure.

The occurrence of diarrhoea towards the end of the course of

treatment may be a sign of emetine toxicity and should not necessarily be attributed to a relapse of dysentery. Once the liver condition is brought under control treatment of any underlying intestinal infection should be instituted. For details of such treatment the reader is referred to standard text books of tropical medicine.

ATYPICAL INTESTINAL AMOEBIASIS—This may present as an emergency in two forms—

Amœbic typhlitis

This because it causes pain, tenderness and thickening in the right iliac fossa resembles appendicitis but the symptoms are usually less urgent and *E. histolytica* may be found in the stool. When operation is not imperative the effect of a few daily injections of emetine should be observed.

Amœboma

An amœbic granuloma may present with signs of intestinal obstruction and may suggest a primary neoplasm. Whenever the possibility of amœbiasis exists in a patient with a tumour of the bowel the effect of emetine should be tried in addition to any other measures that may be indicated.

CHOLERA

There may be a few days of premonitory diarrhœa or the onset may be sudden with rapidly increasing diarrhœa and vomiting. The stools soon lose their feculent character and become colourless and watery. They may amount to several litres a day while profuse vomiting increases the fluid and chloride loss. The viscosity and specific gravity of the blood and its hæmoglobin cell content and hæmatocrit value all rise its alkali reserve falls.

Thirst and restlessness become extreme and there are muscular cramps. The features are sunken and the skin cold, wrinkled and cyanosed. The voice is feeble and the pulse almost imperceptible. The combination of a highly viscid blood with a toxæmia acting on the heart and kidneys finally leads to circulatory failure, anuria and death.

DIAGNOSIS.—While all cases of diarrhœa in a cholera epidemic should be suspect the clinical features mentioned above and the clear watery stools containing flecks of mucus are often sufficient to make a diagnosis even in sporadic cases. Vibrios may be found

on direct examination of the mucus in the stool or after culture for some hours in alkaline peptone water

Treatment—The most important indication is to replace the fluid and salt loss. Clinical manifestations of this are dehydration and collapse muscular cramps oliguria and a systolic blood pressure below 70 mm Hg

Other signs are a rising hæmoglobin hæmatocrit reading and specific gravity of the blood

The latter (normally below 1038) may be readily determined by observing the rise and fall of drops of blood in a series of solutions of copper sulphate of known specific gravity. Whole blood or oxalated blood (not more than 1 mgm of oxalate per c cm of blood) is released from a syringe and needle whose point is about 1 cm above the surface of the copper solution. The drop breaks through the surface of the solution and penetrates 2 or 3 cm below losing its momentum within five seconds after which it begins to rise becomes stationary or continues to fall. The specific gravity of the drop relative to the solution does not change appreciably until the drop has been immersed for 10 to 15 seconds and behaviour during the 10 seconds after it has lost momentum indicates whether it is heavier or lighter than the test solution. A control test using normal blood should be carried out simultaneously

Physiological saline with 5 per cent dextrose should be given by intravenous drip plasma may also be used. In children it may be more feasible to use the subcutaneous intramuscular intraperitoneal or bone marrow route. If cramps are severe hypertonic saline (NaCl 13.75 gm CaCl₂ 0.25 gm water 1 000 c cm) should be used while if there is acidosis or urinary suppression a preliminary alkaline infusion (NaCl 5.75 gm NaHCO₃ 18.25 gm water 1 000 c cm) may be given. If laboratory help is available the amount of alkali needed can be determined as shown on page 8

If the rectal temperature is raised intravenous solutions should be given at a temperature of 80°F so as to avoid hyperpyrexia. The object of infusions is to restore fluid loss to maintain the systolic blood pressure above 70 mm Hg and the specific gravity of the blood below 1060

ADDITIONAL MEASURES—Hyperpyrexia should be controlled by cool sponging. Vomiting is sometimes amenable to sedatives e.g. sodium phenobarbitone gr 1 intramuscularly. A suspension of kaolin and activated carbon 30 gr of each should be given to minimise toxic absorption. Potassium permanganate gr 4 in capsules at 15 minute intervals for two hours with copious drinks may be used to destroy toxins in the gut. Specific anti serum 70

to 80 c cm intraperitoneally has been recommended. Benefit has also been claimed from the use of sulphaguanidine by mouth 6 to 8 gm initially followed by 3 to 4 gm four hourly.

HELMINTHIASIS

Certain urgent situations may be associated with the presence of worms in the bowel or tissues of the host.

Acute allergic manifestations may appear such as are classically associated with rupture of a hydatid cyst. Adrenaline hydrochloride 0.5 c cm of a 1 in 1000 solution should be injected subcutaneously and ephedrine hydrochloride gr $\frac{1}{2}$ or benadryl 50 mgm (gr $\frac{3}{4}$) given by mouth.

Acute mechanical accidents may be caused by worms or the pathological states which they produce. Round worms (*ascaris* (*n. bricoides*)) may for example become impacted in the appendix or bile ducts; they may cause laryngeal spasm during migration while large masses of them are an occasional cause of intestinal obstruction. Intussusception may complicate polyposis associated with intestinal schistosomiasis. In all these conditions appropriate medical treatment should be started once the mechanical defect has been corrected.

Loa loa, a filarial worm may during its migrations appear superficially under the skin, mucous membrane or conjunctiva. It then not only causes a local reaction and much distress to the patient but also presents an opportunity for its removal. When disturbed or cooled these worms migrate rapidly to deeper tissues. If not lying in a position entirely favourable for removal they may sometimes be coaxed to the surface by applying warm compresses. The adult worms resemble stout pieces of thread about two inches long.

After quickly injecting some warmed 2 per cent procaine or instilling some warmed 2 per cent cocaine with adrenaline 0.005 per cent into the conjunctiva the parasite should be seized with forceps and a stitch passed round it and tied to prevent its escape. It may then be gently extracted through suitable incisions. Care should be taken not to tear the parasite lest a severe local allergic reaction result. The removal of the worm is always gratifying both to patient and physician but it should be pointed out to the

patient that filarial infections are more often multiple than single and consequently the symptoms may recur. A course of anti-monial drugs may kill any remaining filariæ but results are doubtful the effect is probably on the adult worms and embryos may remain in the circulation for months.

Tapeworms

The passage of tapeworm segments in the stool demand immediate attention. Gravid segments contain masses of eggs. If

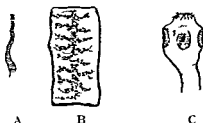


FIG. 25

A and B Head and *nat re* segment of *T sol u n* Natural size Note only 8-12 lateral uterine branches
C Head of *T sol m* $\times 20$ Note double row of hooklets

these are ingested by a suitable host they hatch larvæ which becoming widely distributed form cysticerci. In the central nervous system these may cause epilepsy for which there is no adequate treatment.

Of the common tapeworms of man *Taenia saginata* (the beef tape worm) is unimportant in this respect as its eggs cannot form cysticerci in man. The eggs of *Taenia solium* (the pork tape worm) however will form cysticerci in man as well as in the pig. Hence a patient harbouring *T solium* is exposed to special danger (from regurgitation of eggs or auto-infection) and is a danger to others.

It is important therefore to be able to distinguish the two worms. This is most easily done by pressing a mature segment between two slides and examining with a hand lens. Mature segments are longer than broad. The uterus is easily seen because of the brownish eggs it contains. It consists of a central tube with lateral branches—the number 8 to 12 in *T sol u m*.

whereas in *T. saginata* there are 16 or more (Figs 25 and 26)

The doctor should be careful not to infect himself. Should *T. solium* be found the patient should be warned of the danger of auto infection.

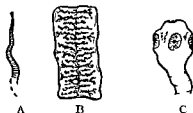


FIG. 26

A and B Head and mature segment of *T. saginata* Natural size. Note 16 or more lateral uterine branches (cf *T. solium*). Mature segments are longer than broad. C Head of *T. saginata* $\times 20$

Treatment—As the patient is in danger so long as *T. solium* remains in the bowel removal of the worm is a relatively urgent matter. The same treatment is used for *T. saginata*. Detailed instructions should be given as follows—

FIRST DAY—Ordinary diet except for omission of vegetables and other high residue foods. In the evening a full dose of cascara (Elixir of Cascara B.P. two teaspoonfuls) should be taken.

SECOND DAY—Fluids and easily assimilable carbohydrates only.

THIRD DAY—Admit to hospital. While still fasting give Extract of Male Fern 6 c cm (90 minims) in three divided doses either as a suspension or in capsules at half hour intervals followed an hour later by half an ounce of Epsom salt and a pint of hot tea. Ordinary diet can be resumed two hours after the saline purgative has acted.

All stools passed for 48 hours after the anthelmintic has been given should be passed into a bed pan and examined for the head of the worm (Figs 25 and 26). This is most easily found if the stool is searched thumbful by thumbful under a gentle stream of water against a dark background such as a slate post mortem room table.

FULMINATING CARDIAC BERI BERI

Acute congestive cardiac failure may arise in the course of a frank attack of beri beri or it may be the first noticed manifestation. As it responds well to specific treatment but may be otherwise fatal it is important to recognise and treat it without delay.

In those predisposed by previous dietetic deficiency the acute attack is often precipitated by strenuous muscular work and seems to be more prevalent in hot weather. Sometimes premonitory symptoms have been present for a few days. The patient becomes dyspnoeic and cyanosed and has epigastric discomfort. Vomiting may occur. Hoarseness caused by pressure on the recurrent laryngeal nerve by the dilated left auricle is sometimes a symptom. The heart is grossly enlarged, the pulse rapid and the blood pressure low. The neck veins are distended and the liver is enlarged, tender and pulsating. Oedema of the subcutaneous tissues and lungs and effusions into the peritoneal, pleural and pericardial cavities are common findings. The urine is scanty and contains albumin and casts.

DIAGNOSIS—The habits of the patient may be a guide to the possibility of beri beri and there will usually be some signs of peripheral neuritis although these may be minimal. Other causes of cardiac failure must be reviewed since in the circumstances in which beri beri occurs mixed factors may well be operative. If beri beri is a possible cause of cardiac failure specific treatment should be started. Where facilities exist helpful tests are those showing a diminished urinary excretion of thiamin and an increased blood content of pyruvate or bisulphate binding substances.

Treatment—Aneurine hydrochloride 50 mgm ($\frac{3}{4}$ grain) should be given intravenously at once. Following the injection the patient may become restless and may not improve for an hour or so. In other cases the response is dramatic. The injection should be repeated eight hourly until it is clear that the response is satisfactory and then a daily dose of 5 mgm (gr $\frac{1}{12}$) continued. Rest and the ancillary measures for cardiac failure are necessary and a generous mixed diet should be ordered supplemented by the B complex of vitamins (e.g. three yeast tablets four times a day).

RABIES

This disease does not present an urgent problem once the symptoms have declared themselves since it is then invariably fatal. The only treatment required is the administration of sedatives *e.g.* sodium phenobarbitone gr 2 intravenously as



FIG. 27

The bloody drooling and facial expression accompanying the pharyngeal spasm of rabies. The hand on the throat is characteristic.

often as necessary to control the distressing symptoms (Figs 27 and 28)

The bite of a possibly rabid animal however always raises the urgent question of prophylactic treatment for the victim. It should be remembered that not only dogs but many other animals including cats, wolves, jackals and vampire bats may act as vectors of the virus. Infection may result not only from biting but also from their licking fresh wounds or abraded skin. Attendants may similarly be infected by patients.

After being absent from Britain for many years rabies has recently reappeared. As it is present on the Continent further cases may be expected if quarantine regulations are infringed.

MANAGEMENT OF SUSPECTED RABIES

Immediate treatment of the wound—Encourage free bleeding by applying a tourniquet just tight enough to impede venous return. Wash the wound with strong potassium permanganate



FIG. 28

The cry and accompanying pharyngeal spasm of rabies

solution. Protect the surrounding skin with vaseline and carefully cauterise the wound with fuming nitric acid or pure phenol; a general anæsthetic may be necessary. The wound should not be sutured for three days, particularly if on the face, head or neck.

Specific treatment—Since this is not entirely innocuous (very rarely there are neurotoxic sequelæ) the important question to answer is—Is the dog rabid?

A rabid dog shows changes in disposition becoming either morose or irritable. The bark changes. The dog is easily startled and appears ill later becoming subject to convulsions or paralysis. Advice may be obtained from the Ministry of Health.

A rabid dog is infective for not more than four days before it develops symptoms after which it dies within six days. When possible therefore a suspected animal should be caged and observed and if it survives 10 days it could not have been rabid at the time of biting. Specific treatment for the victim of its bite would not be required or if commenced could be safely discontinued.

If the animal cannot be caught and observed or if it has been killed and there is a strong reason to suspect rabies or if the wound is on the head where nerve paths to the brain are short it may be advisable to start treatment at once. In the case of peripheral wounds since the incubation period varies from two weeks to several months depending on the length of peripheral nerve to be traversed by the virus there may be time to confirm the possibility of infection before starting treatment.

Treatment consists in giving a series of injections of vaccine prepared from attenuated living fixed virus or killed carbolised or etherised virus. The amount and spacing of doses depends upon the type of vaccine used and the probable severity of the infection. In mild cases 2 c cm of a 1 per cent suspension of infected sheep brain in 0.5 per cent carbol saline may be injected subcutaneously daily for seven days. In cases of average severity 5 c cm may be given daily for 14 days. In the case of severe lacerated wounds on the neck or face 10 c cm should be given daily for 14 days. If vaccine is required application should be made to the Public Health Authority.

Since dogs may carry tetanus spores in the alimentary canal it is a wise precaution to give tetanus anti-toxin 3 000 units to any person bitten by a dog.

SNAKE BITE

Following the bite of a poisonous snake or of any doubtful snake first aid measures should be applied immediately because a lethal absorption of venom may occur within a few minutes although little immediate effect may be observed.

DIAGNOSIS—At the site of a bite of a poisonous snake (Figs 29 and 30) the marks of the fangs may be seen, with bites of colubrine snakes (*e g*, cobra, coral snakes, kraits, etc.), these are not always visible, and the local effects may be trivial

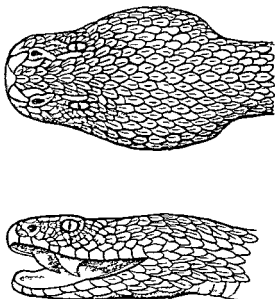


FIG 29

Viperine head—dorsal and lateral views
Note vertical pupil many small scales and
well-defined neck

Colubrine venom contains a neurotoxin which causes muscular weakness, ataxia, dysarthria and diplopia, and later dysphagia, slow shallow breathing and increasing weakness of the voluntary muscles. Convulsions, coma, and death may follow.

The venoms of viperine snakes (*e g*, vipers, rattlers, moccasins, copper heads, fer de lance, etc.) contain proteolytic enzymes which cause a severe local reaction with pain, swelling, discoloration and oozing of blood. Within minutes to hours, general symptoms such as nausea, vomiting and faintness appear, and may progress to cardiovascular failure, hæmoptysis and hæmaturia may also occur. Sepsis and secondary hæmorrhage may be complications in patients who survive long enough.

Treatment—1 Place a tourniquet round the finger, or leg

above the knee or the arm above the elbow—i.e. over a proximal single bone and of course on the heart side of the bite. It should be applied immediately over the clothing if necessary and be tight enough to obstruct arterial flow.

2. Wash the bitten surface with water, urine or saliva.

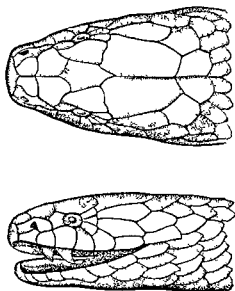


FIG. 30
Coluber c. levi—dorsal and lateral views

3. If possible excise the bitten area immediately. Otherwise incise it, remove any broken fangs and apply mechanical suction which may be kept up for half an hour or so using a breast pump or preheated bottle. The mouth may be used but not if the lips are cracked.

4. In addition put on a venous ligature (i.e. obstructing the veins only) distal to the arterial one. Incise a vein draining the area of the bite and undo the arterial ligature for 30 seconds so that blood enters the part and escaping from the incised vein washes out absorbed venom. Tighten the arterial ligature again and gently milk the limb distally and proximally towards the bite thus squeezing out blood and venom. Release the arterial ligature again for 30 seconds and repeat the whole process.

several times until in the case of the adult some 600 c cm of blood have been removed

5 Liberal amounts of anti-venine should be injected intravenously as soon as possible into an unaffected arm or leg (*For sources of supply in different countries see page 447*) It should also be infiltrated into the bitten area As anti-venines are prepared from horses anaphylactic symptoms may be encountered

6 The affected part should be kept at rest and the patient should not be allowed to walk about Because of the risk of respiratory failure morphine is contra-indicated Artificial respiration and treatment for circulatory failure may be needed

Spitting snakes

Certain colubrine snakes in Africa are able to eject their venom considerable distances and to aim it accurately The patient usually sees the snake and almost immediately feels an intense burning in the eyes Severe conjunctivitis results Treatment consists of repeated irrigation with anti-venine if available or with saline or 1 in 5 000 potassium permanganate and instillation of 1 per cent atropine drops

OTHER BITES AND STINGS

The bite of the "**Black Widow**" spider of America and related species in the Mediterranean zone Australia South Africa and Russia is capable of causing severe symptoms As the eggs are often laid around privies a common site for the bite is the perineum or genitalia The bite may not be felt but two small red spots are to be seen Within 10 minutes of the bite cramping pain all over the body results the muscles become rigid and an acute abdomen may be simulated Other features are pyrexia leucocytosis and a macular rash

Treatment—Morphine may be required Specific anti-venine should be used if available (Mulford Laboratories Philadelphia) Failing this intravenous calcium gluconate 10 to 20 c cm of 10 per cent solution may be used

The scorpion (Fig 31) is an arachnid about 50 mm long with an armoured tail ending in a large sting the poison of which causes burning pain followed later by salivation nausea

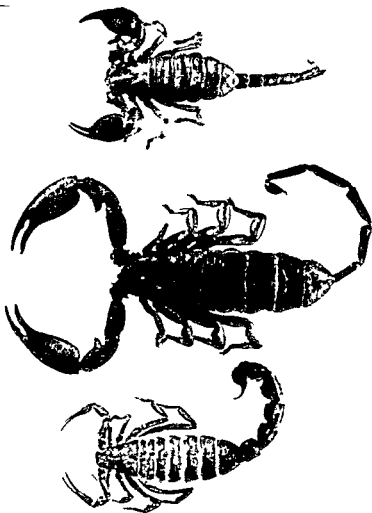


FIG. 31

South African scorpions

(B. J. S. et al. of Tropical Medicine)

vomiting drowsiness and collapse Muscular cramps may be a feature In young children cardio-respiratory paralysis may result in death

Treatment—A tourniquet should be applied and suction started Application of ammonia may relieve the pain Failing this 2 per cent procaine may be injected or opiates may be given Specific anti-venine is available (Allen & Hanbury Ltd 7 Vere Street London W 1 Telephone MAYfair 2216)

Bee wasp and hornet stings

While bee stings are trivial to the experienced apiarist they may be serious to others Occasionally coma and death have followed within 20 minutes Since in these cases no local reaction has occurred and the site has been on thin vascular skin it is assumed that the poison enters a venule by chance Status asthmaticus has followed a sting

Treatment—The sting if still visible should be removed by scraping it out with a knife or a finger nail It should not be picked out with the fingers as this expresses more venom Alkali *e.g.* ammonia blue bag bicarbonate of soda or soap may be applied to bee stings Weak acids *e.g.* vinegar are said to be better for wasp stings

Adrenaline is necessary for the more serious symptoms and part of the equipment of a bee keeper particularly if he happens to be a doctor might well be adrenaline hydrochloride in tubulin ampoules They are supplied by Roche Products Ltd in two sizes containing gr $\frac{1}{200}$ (m 5) and gr $\frac{1}{100}$ (m 10) of adrenaline hydrochloride respectively

Bee-keepers who know they react badly to stings may prevent serious symptoms by taking ephedrine gr $\frac{1}{4}$ by mouth before handling bees Desensitisation by injections of filtered extracts of whole bees has been suggested

Blistering beetles produce skin lesions requiring local treatment by alkaline lotions Fluid from crushed beetles if it reaches the eyes causes an acute conjunctivitis as also do the hairs of various caterpillars Treatment is on general lines

THE EFFECTS OF HEAT

When atmospheric conditions are such that the body progressively gains heat or in an attempt to maintain thermal equilibrium by severe sweating loses chloride and water excessively

an acute physiological breakdown may occur. The clinical manifestations of this are —

- 1 Heat cramps
- 2 Heat exhaustion
- 3 Heat stroke

Heat cramps

These occur in tropical climates or under industrial conditions where the combination of high temperature heavy muscular work and large intake of water causes profuse sweating and loss of salt. This salt depletion leads to electrolytic changes in the muscles particularly of the extremities and the abdomen. Painful and disabling muscle spasms result. Unacclimatised stokers in tropical climates are particularly apt to suffer.

DIAGNOSIS — This is usually easy but when abdominal pain is severe other causes must be considered. Confirmatory evidence is provided by the diminution or absence of chlorides in the urine. They may be tested for as follows —

Filter if not already clear and if albumin is present remove by boiling and filtering. Add to half an inch of urine in a test tube a few drops of nitric acid (pure) and then as much 3 per cent silver nitrate as there is urine. If the chloride content is normal an abundant curdy precipitate occurs at once. If chlorides are diminished the solution merely becomes milky. A trace of chloride causes only an opalescence. In the absence of chlorides the solution remains clear.

Treatment — In severe cases morphine may be needed. Normal saline should then be given intravenously. In milder cases sodium chloride solution (0.5 gm in 500 c cm or one pint of water) may be given by mouth.

A more palatable drink is made as follows. To 3 litres (6½ pints) of water add —

Sodium chloride	1 lb
Potassium chloride	10½ ozs
Citric acid	5½ ozs
Saccharin	$\frac{1}{8}$ oz
Oil of Lemon	$\frac{2}{3}$ fl oz

A little lemon squash colouring and dilute to 20 galls

Heat exhaustion

This includes simple circulatory inadequacy arising from the increased circulatory effort and peripheral vaso dilatation necessary

to maintain heat loss. It also embraces the more serious states of salt deficiency and dehydration from excessive sweating. Skin conditions such as prickly heat which interfere with normal sweating play their part in the breakdown of the defence against heat. Fatigue, alcoholic excess and poor general physique play a part also.

The patient is pale and often has a low blood pressure. Sweating continues (except in the dysidrotic type) and the skin may be cold and clammy. The condition precedes failure of the heat regulating centres and so the rectal temperature is not grossly raised. Heat cramps may complicate the picture.

Treatment —In simple heat exhaustion place the patient in a cool, shady, well ventilated place, loosen the clothing and give simple stimulants such as sal volatile. Leptazol 2 c cm intravenously or intramuscularly may be used. Salted drinks should be given to those suffering from salt depletion. For the dysidrotic type water is chiefly required but the response to treatment is poor and invaliding to a cooler climate is usually necessary.

Heatstroke

This is a serious condition due to profound failure of the heat regulating mechanism. It is characterised by progressive hyperthermia, circulatory collapse, convulsions and coma. Without adequate treatment it is usually fatal. Even after recovery impairment of the heat regulating mechanism may result in inability to withstand heat.

Prolonged exposure to high temperatures is the cause. High humidity preventing heat loss by the evaporation of sweat contributes. Other predisposing factors are atropine (by inhibiting sweating), alcohol (by increased heat production) and anaesthesia (possibly from adrenal stimulation).

Premonitory signs are lassitude, headache, vertigo, nausea and diminution of sweating. The attack may be ushered in by vomiting, muscular twitchings and convulsions. The skin is hot and dry and the temperature and pulse rate raised. There may be diarrhoea and oliguria with albuminuria. Later delirium, drowsiness or excitement give place to coma and death.

Diagnosis —The above features should make this clear but it must not be forgotten that hyperthermia may be the presenting sign in an infective condition such as malaria or pneumonia. Coma

from endogenous and exogenous toxins is not accompanied by hyperthermia. In pontine hæmorrhage coma precedes hyperthermia and if the patient is not seen early diagnosis may be difficult.

Treatment—Whatever the cause of hyperpyrexia suitable measures must be taken at once to restore the temperature to physiologically tolerable limits. If there is any possibility of cerebral malaria quinine should be injected (*cf.* page 284). Cold sponging and continuous fanning should be used until the rectal temperature shows that the condition is under control. Cooling should be discontinued when the rectal temperature falls below 103°F. Ideally the patient should be put in an air conditioned room at 65°F and at a low humidity. Venesection should be done if the blood pressure is raised and the pulse full. Lumbar puncture may help patients with cerebral irritation. Morphine should be avoided. Dehydration may call for intravenous *infusion of physiological saline solution*. *There is a considerable tendency to relapse and so the convalescent patient should remain under careful supervision in a cool environment for a few weeks.*

Patients whose sweating mechanism is inadequate should be excluded from conditions conducive to heatstroke. They may be detected by asking them to paddle up to the knees in a bath of water as warm as tolerable for 5 to 15 minutes in a cool room. The flow of blood so warmed in the legs will stimulate a normal person to sweat sensibly over the rest of the body. An abnormal person will show little or no visible sweat.

FREDERIC MURGATROYD

CHAPTER XIX

Emergencies in Industrial Medicine

THE medical hazards of industry are numerous and may be classified as follows —

PHYSICAL (These are dealt with in other chapters on the pages indicated)

Temperature	Heat cramps	(page 304)
	Heat exhaustion	(page 304)
	Heat stroke	(page 305)
Pressure	Compressed air illness	(page 337)
Electric shock		(page 88)

CHEMICAL

- (1) Asphyxiants, causing oxygen deficiency without direct injury to respiratory tract
 - (a) simple—inert gases displacing oxygen from the respiratory tract *e g* nitrogen carbon dioxide nitrous oxide
 - (b) chemical interfering with the vascular respiratory mechanism *e g* carbon monoxide hydrogen cyanide and allied products aniline nitrobenzene and other nitro and amido derivatives of benzene
- (2) Irritants, causing inflammation of respiratory tract and lungs
 - (a) acting on upper respiratory tract *e g* ammonia hydrochloric acid and sulphur dioxide
 - (b) acting on upper respiratory tract and lungs *e g* sulphur dioxide chlorine and other halogens
 - (c) acting principally on lungs *e g* nitrous fumes phosgene arsenic trichloride nickel carbonyl

- (3) Centrally acting poisons producing anæsthetic effects after absorption through the lungs Volatile solvents—
- (a) hydrocarbon group *e g*, benzene toluene, petrol benzine, white spirit, and solvent naphthas
 - (b) chloro compounds *e g* tri chloroethylene carbon tetra chloride methylene dichloride methyl chloride methyl bromide chloroform
 - (c) alcohols *e g* methyl alcohol
 - (d) miscellaneous *e g* organic nitrogen compounds acting upon the blood and circulation (nitrite effect) amyl nitrite
- (4) Miscellaneous poisons Lead and lead tetra ethyl arsenic uretted hydrogen hydrogen sulphide carbon bisulphide nicotine phenol metallic particles producing metal fume fever

(For poisoning on land ship see page 335)

Poisonous substances may enter the body in three ways —

- (1) VIA THE LUNGS This is by far the most important portal of entry Inorganic dusts the fumes or dusts of metals the vapours of organic solvents and most toxic gases are absorbed in this way
- (2) VIA THE SKIN This route is less common but important to recognise because of the necessity of removing contaminated clothing at the earliest opportunity The noxious substances must be removed by washing or other means Tetra ethyl lead aniline nitrobenzene phenol cyanide and nicotine can be readily absorbed through the skin and rapid poisoning result It must

also be remembered that liquids splashed on the clothes (e.g. liquid phosgene) will vaporise when the patient is taken indoors and aggravate his symptoms by being inhaled.

- (3) VIA THE GASTRO INTESTINAL TRACT This is comparatively unimportant in industry but applies to accidents in laboratories.

If a person is taken suddenly ill while at work it is usually possible to obtain an accurate and detailed history of his precise occupation and to assess the results of exposure to toxic substances. Works managers, chemists and engineers are becoming increasingly aware of the symptoms of industrial poisoning but it must be emphasised that accurate information regarding the patient's occupation is indispensable in establishing the correct diagnosis. It is essential to know the materials to which the patient has been exposed and the doctor should insist on interviewing the factory manager or other responsible official. If the patient is seen away from his place of work (and many cases of occupational poisoning arise some hours after leaving work) a direct approach should be made to the factory concerned. In doubtful cases the help of the Medical Inspector of Factories should be sought.

The common emergencies arising from occupational poisoning are asphyxia, coma and convulsions. Less serious symptoms are pulmonary irritation and a variety of symptoms arising from interference with the functions of the central nervous system. A few industrial poisons produce characteristic clinical pictures but in the main the symptoms do not differ from those which may arise from acute illness of non-occupational origin. Even with a complete history and details of exposure non-industrial illness has to be excluded. The principles of treatment are well established and few cases of occupational poisoning require specific antidotes.

GENERAL PRINCIPLES OF TREATMENT—On receiving an emergency call to an industrial establishment the doctor will usually find that the first indication is for the symptomatic treatment of asphyxia and coma. First aid measures will have been adopted

as a rule efficiently because of the increasing attention given to this service in industry

Coma and convulsions should be dealt with on the lines indicated on *pages 148 and 163*

ASPHYXIA

Asphyxia is commonly seen as a medical emergency in industry associated with the effects of the materials classified on *page 307*. It may be assumed to be present in all cases showing cyanosis as well as in all cases of carbon monoxide poisoning. When coma is the presenting sign there is almost invariably some degree of asphyxia and death results from respiratory failure.

RESCUE—When called to casualties following an escape of gas the following principles of rescue must be observed

- (1) Keep to the windward side of the gas leakage and warn people in the vicinity
- (2) Put on a breathing apparatus (*page 393*)
- (3) Shut off the source of gas if this is possible
- (4) Open all doors and windows and leave the building until it is clear of gas

After rescue it is essential to keep the patient at rest even if he is only slightly affected and no exercise must be permitted for some hours after recovery. After removal of the patient from contact with the poisonous material which may include stripping and washing him the doctor should adopt the following principles of treatment

Treatment—Remove the patient to a pure atmosphere and lay him down with face to windward. Rescuers must under no circumstances walk the patient about

- (1) Ensure that he has an adequate airway and that this is maintained during treatment
- (2) Commence artificial respiration if breathing has ceased. Methods of performing artificial respiration are discussed on *page 395* but a few special points require mention. Schafer's method is taught widely in industry and where special risks obtain apparatus is commonly provided for Eve's rocking method. The doctor should stress that success depends largely on the speed of commencing operations and the efficiency of the operator. Many cases of industrial

poisoning require very prolonged artificial respiration and the doctor should see that this is carried out

- (3) Remove or cut away contaminated clothing (*e g* splashed with HCN)
- (4) Keep the patient warm with blankets and hot water bottles
- (5) Administer oxygen or oxygen carbon dioxide mixture (7 per cent CO_2 and 93 per cent O_2) This is unquestionably the most important single measure in the treatment of acute medical emergencies in industry. Many industrial concerns have supplies of oxygen and masks available. The mixture of oxygen and carbon dioxide is suitable for all cases of emergency and may be administered by a portable apparatus [The Novita Seibe Gorman & Co Ltd (*page 403*) or a B L B mask (*page 411*)]
- (6) Circulatory failure may call for the use of nikethamide 1 to 2 c cm or leptazol 1 to 2 c cm by intramuscular injection. It is almost invariably secondary to asphyxia however and its effective treatment depends upon the relief of the asphyxia as already described.

Venesection should be practised only if there is evidence of embarrassment of the right side of the heart such as marked engorgement or pulsation of the jugular veins and not for cyanosis alone.

Having dealt with the immediate emergency measures more detailed methods of dealing with individual poisons may be considered.

ASPHYXIANTS

All users of asphyxiant products must be acquainted with the first aid measures required in the event of an accident and resuscitation and rescue apparatus should always be at hand. Workers should be practised in the use of the equipment and in the Schafer method of artificial respiration. The action of many of the compounds in this group (HCN in particular) in high concentrations is so sudden that a man entering a contaminated atmosphere may collapse before he has time to act on any warning he may receive.

Simple asphyxiants are inert gases which cause no direct injury to the respiratory tract or internal organs but produce oxygen deficiency and hence asphyxia. Carbon dioxide is a good example and its effects are encountered in a variety of places.

e.g. breweries and mineral water works lime kilns and ship-holds The treatment of simple asphyxia has been described

Of the chemical asphyxiants **carbon monoxide** is the most important and is responsible for between one half and one third of all reported gassing accidents in factories in Great Britain The risk exists in many industries particularly in those with gas producers and blast furnaces but exposure to coal gas and motor exhaust fumes occurs in all spheres of life Having no colour vapour or odour CO is rapidly absorbed via the lungs and is a particularly dangerous substance Entry into high concentrations produces almost instantaneous loss of consciousness For treatment *see page 4*

Cyanides are encountered in many chemical industries and are used in dyeing the manufacture of plastics and for fumigation and the hardening of metals

Hydrogen cyanide is highly toxic colourless and volatile having an odour resembling that of bitter almonds which may not be noticeable however even in high concentrations It probably acts directly on the body cells interfering with normal oxidation The respiratory centre is particularly sensitive to its action which accounts for the early respiratory paralysis

In industry poisoning results rarely from swallowing solid or liquid cyanide It more commonly arises from the inhalation of hydrocyanic acid gas (HCN)

Owing to the high volatility of HCN the danger of spilling the liquid on the bare skin appears to be slight as long as evaporation is unimpeded

The action of HCN in high concentrations is so sudden that a man entering an atmosphere containing it may collapse at once It is important therefore that when the presence of HCN in the atmosphere is suspected a benzidine copper acetate test paper should be exposed before the room is entered It is prepared by saturating a strip of absorbent paper with a solution freshly made by mixing equal parts of an aqueous solution containing three grams copper acetate per litre and an aqueous solution containing one gram of benzidine acetate per litre This is exposed for at least 10 seconds If HCN is present it turns blue

SYMPTOMS OF POISON —The early warning symptoms are a sensation of irritation in the throat increasing difficulty in

breathing lachrymation followed by headache dizziness nausea vomiting and general weakness with a feeling of heaviness in the arms and legs

At the first sign of any of these symptoms a person must immediately leave the dangerous atmosphere. Warning notices should be immediately affixed in the area and the test carried out. Thereafter entry is at the discretion of a responsible member of the technical staff who will naturally supervise all further investigations and safety measures.

Signs of severe poisoning are pallor and increasing shock unconsciousness and cessation of breathing. There may be tetany and convulsions and signs of cardiac failure.

FIRST AID MEASURES—When the patient has been rescued and the contaminated clothing removed or cut away break a capsule of amyl nitrite and allow the patient to inhale the vapour. Immediately proceed with artificial respiration and the administration of oxygen. If cyanide comes into contact with the skin the affected area should be repeatedly washed with water.

The modern treatment of cyanide poisoning is based on the administration of nitrites which convert cyanide in the blood into the non toxic cyanmethæmoglobin. It is believed that the nitrite combines with some of the available hæmoglobin to form methæmoglobin which then combines with cyanide to form cyanmethæmoglobin. Amyl nitrite is readily absorbed so long as the patient is breathing and is stated to form methæmoglobin in 10 to 15 seconds.

Following rescue and first aid measures the following treatment is advised—

- (1) Continue with artificial respiration if possible using Eve's rocking method
- (2) Administer nikethamide 1 to 2 c cm intramuscularly and/or lobeline hydrochloride 1 c cm ($\frac{1}{6}$ gr) subcutaneously
- (3) Repeat amyl nitrite inhalations for three to five minutes
- (4) Inject a solution of sodium nitrite 0.3 gm in 10 c cm of water intravenously at a rate of 2 to 5 c cm per minute
- (5) Inject intravenously through the same needle 25 to 50 c cm of a 50 per cent solution of sodium thiosulphate at the same rate. This increases the efficacy of the treatment by converting cyanmethæmoglobin into a relatively harmless and easily excreted thiocyanate.

These procedures may be repeated, in half dose, an hour later if symptoms recur. It is necessary to observe the patient closely for 24 to 48 hours.

Aniline and the nitro and ammo derivatives of benzene.

These produce asphyxia and nervous symptoms such as headache, vertigo, nausea and vomiting. There may also be irritability, somnolence, or mental confusion associated with an unsteady gait, muscular tremors, and finally convulsions and coma. It is especially important to remember that absorption takes place by the skin, as well as by the lungs, and that the patient may become affected at home, or on his way home.

He should be immediately stripped, the skin washed with soap and water, and clean clothes put on. Otherwise treatment is symptomatic. The compounds produce pallor and cyanosis by the formation of methæmoglobin in the blood. The grey blue colour of the lips is characteristic.

Mono nitrobenzene (murbane oil), which is used for making shoe and floor polish, produces similar but less acute symptoms to dinitrobenzene. Similar effects are produced by the nitrophenols but, in addition, dinitrophenol has the curious effect of accelerating metabolism, causing loss of weight. Treatment of the effects produced by this group is along general lines and no specific remedies are indicated.

LUNG IRRITANTS

In many operations in industry exposure to lung irritant gases or vapours may occur. Some of these act on the upper respiratory tract causing immediate irritative symptoms, while others have a lesser effect there but damage the lungs and cause serious delayed symptoms. There is little essential difference between the actions of the various members of this group, and the treatment and prognosis depend on the extent of the lesions produced in the lungs. The concentration of gas to which the patient is exposed largely determines the clinical picture.

Hydrochloric and sulphuric acid and ammonia are commonly encountered in industry and cause severe burns although intense irritation of the upper respiratory tract and eyes may occasionally occur without obvious burns. Bronchitis may ensue, but serious complications such as pulmonary œdema are rare.

Chlorine gives rise to immediate irritation of the upper respiratory and lachrymatory tracts and causes considerable respiratory spasm and discomfort. Patients are disabled immediately but recover in a comparatively short space of time and the only after effects observed in industry are laryngitis and bronchitis. In mild cases, the face is flushed the respiratory rate slightly increased, and the cough painful in more severe cases there is considerable respiratory embarrassment with distended veins and cyanosis. Medium rhonchi and râles are heard in the chest.

Sulphur dioxide produces effects similar to those of chlorine in chemical and furnace workers.

Phosgene is used in the chemical and dyestuffs industry and along with **nickel carbonyl**, it has an important delayed effect on the lungs.

Nitrous fumes may be encountered in the manufacture of nitric acid and explosives and also in welding. They cause symptoms less severe than do the irritants previously mentioned. The patient recovers fairly rapidly, and may not appear distressed or ill but delayed pulmonary oedema may occur.

The difficulty in deciding the degree of gassing is notorious for the condition of the patient immediately after the accident is no guide to the ultimate outcome. Absolute rest should be secured at once and the patient observed in hospital for 24 to 48 hours in order to prevent pulmonary oedema or minimise its development.

Treatment of lung irritant gassing—The two essentials are rest and oxygen. The patient should be carried into a pure warm atmosphere free from draught. He must not be permitted to walk. Clothing at the neck and waist should be loosened and any contaminated garments removed. He must be kept at rest though an occasional change of position from lying down to sitting up may be beneficial. He should be reassured and encouraged to suppress his desire to cough.

Warmth (hot water bottles and blankets) should be applied and drinks of hot sweetened tea or coffee given. Dyspnoea or cyanosis should be treated by the administration of oxygen or oxygen carbon dioxide mixture (see page 409). Artificial respiration is not indicated.

Immediate symptoms are more distressing in cases caused by irritants such as chlorine which affect the upper respiratory tract. Relief can be obtained by the inhalation of a steamy atmosphere from a bronchitis kettle or from a vessel containing two teaspoonfuls of Friars' Balsam (compound tincture of benzoin B.P.) in a quart of hot water. The desire to cough can be relieved by the administration of one teaspoonful of the camphorated tincture of diamorphine (B.P.C.) or of the elixir of terpine hydrate with ethylmorphine hydrochloride (B.P.C.). It is not desirable to give morphine and little benefit is to be expected from the use of atropine even in cases where pulmonary oedema may be expected. Venesection should only be practised if there is definite evidence of embarrassment of the right heart and not for cyanosis alone. If circulatory failure is evident injections of nikethamide or leptazol (1 to 2 c.c.) may be beneficial.

THE VOLATILE SOLVENTS

Various solvents are used in industry and numerous cases of intoxication are encountered both in large works and in small premises such as dry cleaning establishments. Generally they produce headache, giddiness and a sense of heaviness of the legs followed by inebriation, drowsiness, loss of consciousness and death from respiratory failure. Many are good anaesthetics and are used for this purpose. Several members of the group have delayed effects on the liver and kidneys. The treatment of casualties is on general lines—rest, oxygen and artificial respiration.

Certain special features of particular solvents will now be considered. It is important to recognise them because many solvents have a heavy vapour and the patient exposed to them must be kept off the floor. Some solvents are inflammable and others are decomposed by heat and it is not permissible to smoke in or near their vapours. A person affected by solvent vapours even if only slightly must in no circumstances be permitted to resume work or exert himself by walking or cycling. He should be taken home by car and instructed to rest for the remainder of the day preferably in bed.

Benzene, Toluene

Exposure to small concentrations causes headache, giddiness, muscular twitchings and excitement. After severe exposure these symptoms may be followed by convulsions, coma and death.

Chloro compounds

Methyl chloride is used as a refrigerant and in addition to the above symptoms causes convulsions and oliguria

Carbon Tetrachloride rarely causes narcosis when encountered in industry but vomiting commonly occurs after exposure and the symptoms of an acute abdomen are occasionally produced by direct action on the small bowel and colon causing spastic contraction. There is sudden onset of epigastric pain and vomiting associated with increasing tenderness in the loins. The urine is scanty and contains red blood cells. Both methyl bromide (see also page 336) and carbon tetrachloride are used in fire extinguishers. In addition to the symptoms of solvent poisoning described methyl bromide produces burns of the skin.

NITRITE EFFECT—Nitro-glycerine is a well known oily explosive liquid which produces methæmoglobin in the blood and causes dilatation of the blood vessels, a fall in blood pressure and slowing of respiration. Severe headache follows the inhalation of vapour but acute poisoning is rare in industry. Amyl nitrite has a similar effect.

The symptoms usually subside satisfactorily if the patient is kept lying down and the effects may be alleviated by injections of strychnine (gr $\frac{1}{16}$ to $\frac{1}{8}$). Artificial respiration may be necessary.

MISCELLANEOUS POISONS

Lead

This constitutes a widespread occupational hazard to workers employed in the extraction, smelting, refining, melting and use of metallic lead and its compounds. Lead is principally absorbed via the respiratory tract following exposure to fumes and dust but absorption also occurs from the gastro intestinal tract and from the skin in the case of tetra ethyl lead.

Lead intoxication indicated by palsy, colic and encephalopathy must be clearly distinguished from lead absorption. The urgent symptoms of colic and encephalopathy must be attributed to lead if a worker exposed to lead shows other evidence such as anæmia, punctate basophilia, a black line on the gums, muscular tremors, paralysis and the presence of lead in the faeces and urine. In lead poisoning the urinary lead concentration is generally

greater than 0.150 mgm per litre the upper limit for normal persons being 0.03 mgm per litre

LEAD COLIC—This is characterised by sudden intense griping pain around or below the umbilicus. The patient is cold, pale and perspiring. The abdomen is tense but no true rigidity is present on examination between the spasms. There is a history of constipation and sometimes of a prodromal period in which there has been anorexia, nausea and intermittent pain in the lower abdomen.

The local application of moist heat and the administration of enemata and magnesium sulphate and belladonna are classical remedies. Immediate relief is usually obtained by the intravenous injection of 15 c.c. of a 20 per cent solution of calcium gluconate or 10 c.c. of a 5 per cent solution of calcium chloride. A similar amount of calcium gluconate may be given intramuscularly and repeated if the pain recurs.

LEAD ENCEPHALOPATHY—This is now almost unknown in this country but has been described following severe and prolonged exposure to inorganic lead and particularly to tetra ethyl lead.

Tetra ethyl lead is absorbed through the intact skin and readily through the pulmonary epithelium. Being highly lipid soluble it accumulates in the nervous system producing acute poisoning. The most important symptoms are attributable to a widespread stimulant action on the central nervous system producing restlessness, trembling, twitching, ataxia, convulsions and coma. Delirium and delusions may occur particularly if the onset is abrupt. Constipation is not a feature of tetra ethyl lead poisoning nor is stippling of the red cells usual. The pulse rate is slow, the temperature subnormal and both the systolic and diastolic blood pressures low. The diagnosis is based on a history of exposure and with the symptoms described may be confirmed by the presence of lead in the faeces and urine.

In treatment it is important to cleanse the skin by washing with spirit followed by soap and water. Other measures are symptomatic, being directed to supplying fluid and ensuring sleep. In severe cases it is necessary to ensure adequate fluid intake by means of glucose and saline intravenously. Delirium may be relieved by 4 to 6 fl. oz. of a saturated solution of magnesium sulphate given as an enema. Hexobarbitone B.P.

gr 4 to 8 or Pentobarbitone B P (Nembutal) gr $1\frac{1}{2}$ to 3 may be given in full doses and repeated as required. Strict nursing supervision is necessary in view of the suicidal tendencies which are sometimes shown.

Metal fume fever

This is a form of acute industrial poisoning which occurs most commonly in brass casters and is known as brass founders' ague. It also occurs in people exposed to finely particulate metals containing zinc and this is the essential cause. The initial symptoms are dryness of the throat and irritation of the respiratory tract with some cough. Headache, fever and chills, pains in the limbs and sweating occur later. The attack usually subsides in 4 to 6 hours and the patient is restored to normal. Treatment is symptomatic and no specific treatment is known or necessary.

Arsenuretted hydrogen (Arsine, AsH_3)

This is produced in industries where metals and acids react and where arsenic is present in one or both of the materials. It is one of the few poisons encountered in industry which may cause sudden death, being quickly absorbed through the lungs, producing rapid and severe hæmolysis. The early symptoms are faintness, weakness and intense headache with shivering and severe abdominal and muscular pains. An early sign is hæmoglobinuria which is followed by jaundice, anæmia and suppression of urine.

Treatment consists in the administration of oxygen, free fluids and alkalis. British anti-lewisite (BAL) has been reported as effective against arsenic (and also against mercury and cadmium). Its use should therefore be considered (*see page 238*). Blood transfusion is indicated where there is severe anæmia and certainly if the hæmoglobin is below 60 per cent or the red cells below 4 000 000 per c mm. It is necessary to perform the transfusion slowly to avoid circulatory overloading (*see page 30*). Liquids and glucose should be given freely by the mouth or rectum. Sodium citrate and sodium bicarbonate should be given in sufficient amounts to maintain an alkaline urine. 20 gr of each every hour until the urine is alkaline and thereafter the same dose four hourly. It is believed that alkalis prevent blockage of kidney tubules by acid hæmatin. Oxygen should be given if there is anæmia since anoxæmia may be present in the absence

of cyanosis. In severe cases death is often preceded by anuria. The patient should be watched until the blood and urine are normal. When damage to the liver, spleen and kidneys occurs it is probably caused by arsenic itself rather than the effects of anoxia and blockage of the kidney tubules.

Carbon bisulphide (CS_2) and hydrogen sulphide (H_2S)

The effects of these substances are similar but hydrogen sulphide produces the more acute symptoms. Exposure may occur in artificial silk workers, tanners, sewer workers and in various chemical operations. Carbon bisulphide is used as a solvent for rubber and is more commonly a cause of chronic poisoning.

Exposure to high concentrations of hydrogen sulphide may cause sudden death but it is well to remember that this occurs but rarely in industry, HCN and arsine being the two other substances having a similar effect.

Absorption is mainly via the lungs but carbon bisulphide is also absorbed by the skin. The essential action of both substances is paralysis of the central nervous system. The symptoms of acute poisoning are intoxication and narcosis, sometimes preceded by headache, vertigo and general weakness. The characteristic smell may be little noticed even in a high concentration of the gas. Mental disturbances such as agitation and hallucinations are common in cases of exposure to H_2S and CS_2 . Even with serious symptoms recovery is the rule and treatment is along general lines, the essentials being the administration of oxygen combined with artificial respiration where necessary. Being a highly diffusible gas with a low molecular weight, H_2S is not readily adsorbed on to activated carbon. The standard respirators therefore offer no protection against the gas.

Nicotine

The use of nicotine in industry is increasing particularly in the manufacture of insecticides. It is absorbed readily through the skin and causes excitation and then paralysis of the central nervous system. The immediate symptoms are nausea, vomiting, sweating and dyspnoea. If a concentrated solution is spilled on the skin it must be removed immediately by scrubbing with soap and water. Artificial respiration may be necessary for a prolonged period.

Phenol

This is absorbed by the skin and lungs and fatal poisoning has resulted from absorption by both routes. Headache, vertigo, nausea, tinnitus, faintness, excitement, convulsions, and respiratory paralysis may occur.

The treatment must be directed towards removing any skin contamination by washing with soap and water. Subsequently oxygen and artificial respiration should be employed.

A. THELWALL JONES

CHAPTER XX

Medical Emergencies at Sea

THE situation confronting the doctor when an urgent medical illness occurs on board ship has always been complicated by environmental factors. Of these dirt and under nourishment have largely been abolished but rough weather seasickness and shipwreck still threaten the sailor. In addition scientific improvements have brought their own dangers and to replace the scurvy the fever and the fluxes of the sixteenth and seventeenth centuries come the fumes the bends the cramps and the blasts of modern times. There has been little fundamental change however in the nature of the mariner and in the restricted world of ship life he thrives on rumour loves gossip and still retains some of the superstitions of the past. Unlike most forms of human society the social structure in a ship has remained feudal all owing allegiance to the master and forming as Dudley says a special herd in a special environment.

The importance of these points to the doctor is to show him how necessary it is that he should combine old wisdom with new knowledge. He must allay apprehensions and inspire confidence as well as know the treatment of diseases met at sea. Discipline too is essential to the working of the ship and he must therefore do nothing that would detract from the authority of the master and the ship's officers. He should take action in emergency if necessary asking the advice of doctors travelling as passengers and if circumstances warrant it he should let it be known that suitable action is being taken. On the other hand elaborate treatment is unsuitable at sea and should not be entertained.

MEDICAL AID TO OTHER SHIPS

Fairly often medical advice is sought by a ship not carrying a doctor. If in convoy the message may be received by visual signal or loud hailer but normally radio transmission is used often allowing direct conversation. The master of the ship seeking advice is instructed to acquaint the doctor with his medical resources and the type of medicine chest carried he has

with him the Ship Captain's Medical Guide and reference to this and the 1931 International Code of Signals (Medical Section Vol 11 pages 233 246) will show how details of a case should be given to the doctor. Whenever possible the doctor should reply with the diagnosis directing the master to the treatment in his book. he should make sure that any drug ordered is actually carried on board. The doctor may also have to give an opinion as to whether the ship should continue on her course or make for the nearest port. Since he cannot examine the patient instructions given by wireless do not involve him in any legal responsibility either through errors in transmission or from any other cause.

When advice is asked about a fever quinine should be ordered if there is the slightest possibility of malaria. Since men collapsing in an engine room in the tropics are very often suffering from cerebral malaria and not heat stroke it is necessary in such cases to give instructions on how to inject quinine.

When ships are close together it may be possible for the doctor to visit the patient by ship's boat. Failing this various devices such as a Coston gun or a buoy have been used for sending drugs.

SEASICKNESS

In spite of innumerable treatments this malady persists and it is fair to say that most passengers will still be sick in small ships in very rough weather. Sufferers react in different ways some like to go on deck while others will not leave their cabins some take glucose others champagne while others put their trust in a tight abdominal binder.

While no treatment is specific hyoscine is well worth a trial one hour before rough weather is expected gr $\frac{1}{100}$ should be taken by mouth, this may be followed at six hourly intervals by gr $\frac{1}{100}$ for 48 hours. It produces in many people a remarkable feeling of stability and seasickness can often be avoided until sea legs are gained. Side effects are dryness of the mouth and drowsiness and sometimes interference with accommodation for near vision.

In severe cases it is wise to be on the lookout for evidence of dehydration and anticipate it by ordering frequent sips of glucose drinks. Very occasionally intravenous dextrose and

saline may be necessary, but however extreme the prostration, death practically never occurs in uncomplicated cases

When symptoms are severe, other conditions may also be present. Thus, when meningitis occurs at sea, an all too common story is that "the doctor first thought it was seasickness." The same is true of diphtheria, when the sore throat has been attributed to persistent retching, with tragic consequences. Less important, but also worth remembering, is that the vomiting of pregnancy and seasickness may occur together.

SHIPWRECK

Even in peace time, problems resulting from shipwreck form some of the most important medical emergencies and the ship's surgeon may be called upon to show the qualities of a seafaring man.

Precautions against shipwreck are the responsibility of the master, but on ship's rounds a tactful enquiry about the water and food capacity of lifeboats may stimulate interest towards improvements. Thus, although on an average each boat carries 40 gallons of water, some masters have managed to improvise stowage for as much as 120 gallons per boat. Further knowledge about actual conditions in lifeboats (location of medical supplies etc.) can be gained by the doctor taking part in exercises when a boat is lowered for a practice pull or sail. He should also make it his personal responsibility to see that a copy of the Medical Research Council War Memorandum No. 8 which deals with the preservation of life at sea after shipwreck, is stowed in each boat. He himself should be thoroughly conversant with the contents of the publication.

Before abandoning ship, everyone should try to take a long drink of water, and collect extra warm clothes. These are essential, even in the tropics, where it can be bitterly cold at night. Wet clothing, particularly socks and boots should be dried as soon as circumstances permit. Theoretically, resuscitating the drowned will be the earliest problem but in practice it may be more important to prevent men who are struggling in the water from upsetting a fully loaded boat.

As water is the most important single factor in determining survival attention must at once be given to rationing supplies. Each boat is scheduled to carry at least 5½ pints per man. If

there is likelihood of being in the boat for say a week the ration recommended is 18 fl oz per man daily. None should be given in the first 24 hours then 6 fl oz thrice daily until one pint per man remains when the ration should be reduced to 2 fl oz per man per day. Alcohol should be reserved for



FIG 37

Immersion foot

Surgery of Modern Warfare

the wounded and moribund for others it will only increase thirst and heat loss. Regular sleep should be obtained during watches off but it may be necessary in periods of emergency for some or all of the boat's company to stay awake. At these times the use of 5 mgm tablets of amphetamine sulphate (benzedrine) or desoxyephedrine hydrochloride (methedrine) is recommended. One or two tablets of each thrice daily may be used.

Treatment after rescue—Above all else survivors need drink, food, warmth and prolonged sleep. For the last phenobarbitone should be given for several days but it should be remembered that after exposure to extreme cold susceptibility to sedatives is increased and smaller doses than usual are effective. A big meal should not be taken for three or four days, bread and

milk or thick soup is recommended to start with. If dehydration is severe a slow glucose saline infusion is better than either blood or plasma both of which increase viscosity.

Survivors are prone to curious hallucinations possibly caused by cerebral anoxia. If troublesome the effect of oxygen should be tried. Certain other diseases often but not exclusively found in survivors and caused by exposure are as follows —

(1) **Immersion foot** — This is particularly liable to occur in men who have stood on a partially submerged raft for several days. The feet become swollen, numb and white and in severe cases ulceration of the skin occurs usually associated with salt water boils (Fig. 32). The muscles are weak and there is anaesthesia of the stocking or slipper type. Pain is marked only during recovery when agonising burning sensations are experienced in the soles of the feet.

Treatment — Little can be done in the way of prevention though if men wear sea boots and keep moving they are less liable to suffer.

When the condition has developed the essential treatment is to keep the patient warm and his feet cold as it is harmful to heat the affected part until the oxygen supply is adequate. Therefore remove the patient's clothing and wrap him in warm blankets do not allow him to walk. The feet and legs should be gently raised on pillows and left exposed to the air. Use an ordinary fan intermittently to play cool air on the feet reducing the temperature to between 70° and 80°F. A bath thermometer wrapped in cotton wool can be used for controlling the treatment. After several days the pain and swelling are lessened and the air temperature can be raised and gentle exercises ordered. Abrasions should be dusted with sulphanilamide powder and protected by light dressings. It is wise to give anti-tetanic serum. Diet should be of a high calorie value and it is probably helpful to give supplementary vitamins.

(2) **Frostbite** — (See page 305)

(3) **Snow blindness** — This results from exposure to ultra violet rays which are largely reflected from snow surfaces. Symptoms develop after a latent period of from four to five hours and consist of burning pain, lachrymation, photophobia, blepharospasm and swelling of the palpebral conjunctiva.

Treatment—Tinted glasses (preferably Crooke's glass) should be used prophylactically. If the condition is established, dark glasses and cold compresses should be ordered. The inflammation subsides within two or three days.

(4) **Sunburn.**—Severe sunburn causes blistering of the skin and when very extensive there is also constitutional disturbance—fever, malaise and headache—(popularly known as "sun stroke") and caused by toxic substances absorbed from the damaged skin. Many cases occur on pleasure cruises despite all warnings. Those with fair skins suffer the most.

In the Services reporting sick was prevented by making the condition a punishable offence. Obviously this is not practicable in civilian life but the habit of sleeping in the sun for long periods should be discouraged and instructions circulated giving safe lengths of time for sunbathing. Red or brown grease paint affords some protection. When insects and sun combine to attack the skin the following lotion is useful in preventing bites—

R	Phenol	m	7
	Thymol	gr	5
	Weak solution of Iodine	m	60
	Camphor water to 1 fl oz		

For established sunburn blisters may be opened under aseptic conditions and the raw areas covered with calamine lotion containing 1 per cent of phenol or 2½ per cent of solution of coal tar. This will allay itching but sedatives such as phenobarbitone gr ½ to 1 may also be necessary to ensure sleep.

THE EFFECTS OF EXCESSIVE HEAT

It is a common event in any ship for a man to collapse at his work or for a passenger to complain of a "turn" "dizzy spell" or "black out". When this happens in hot weather the question of how far the condition may be attributable to heat must be considered. The doctor therefore, should know the symptoms and signs of heat affections—not only in order to treat these conditions but also to avoid the repercussions of unfortunate actions such as stripping and plunging into iced water, influential passengers who are only suffering from the "vapours" in their convalescence from some acute debilitating illness.

The main syndromes are described under "Emergencies in Tropical Medicine" (page 303).

THE MANAGEMENT OF EPIDEMICS

It is impossible to lay down hard and fast rules for handling infectious disease because circumstances vary with the number and type of cases. Passengers can be isolated in spare cabins as far aft as possible while for the ship's crew a hospital is usually provided. In smaller vessels improvised isolation can be arranged by rigging a tent over the after hatch, constructional details being given in the Ship Captain's Medical Guide.

In severe cases it will be necessary to detail one or more members of the crew to act as sick nurses. These men must observe the usual precautions such as wearing special overalls or oilskins while at work and they should come into contact with the rest of the crew as little as possible.

Smallpox

The nurse should either have had the disease or have been recently vaccinated successfully. Everyone on board should be vaccinated or re-vaccinated. Only those who can show documentary evidence of successful vaccination within the preceding 12 months should be excluded and the presence of a scar should be checked. Protection of contacts put ashore at intermediate ports before the discovery of smallpox should not be overlooked. In practice difficulties may arise because enough lymph may not be available or because even when stored in a refrigerator it may lose its potency within 14 days from the date of issue. On arrival in harbour the Port Medical Officer will inspect the vessel and give instructions and help concerning vaccination, the disposal of the sick and the follow up of contacts.

Bacillary dysentery

This is one of the commonest infections occurring on board ship and on vessels bound for the East it often flares up among the crew and spreads to the passengers within a few days of leaving Suez. As in practice ashore these minor epidemics are usually transient and the carrier of the infection rarely identified. Enquiry should be made about recent diarrhoea among the food handlers of the crew and the standard of personal cleanliness of the kitchen staff must be closely scrutinised. Members of the crew who have been ashore at Mediterranean ports and taken food there should be considered suspect. Frequently on these

occasions passengers come forward with suggestions for controlling the epidemic for obvious reasons they should be given a careful and courteous hearing Drinking water bilges rats flies and bugs are invariably indicted Useful information—though lacking any bearing on the epidemic—may thus be gathered Extra cleaning and scouring should be ordered and stringent measures adopted to eliminate flies and vermin It is usually worth while to publish a statement indicating that suitable steps have been taken thus reassuring passengers who may otherwise be apprehensive and lose confidence in the medical arrangements on board and in the efficiency of the crew as a whole (*For treatment of bacillary dysentery and other types of acute diarrhoea see pages 49 and 275*)

Malaria

Suggestions for the prevention diagnosis and treatment by ships surgeons of malaria on ships are given in a pamphlet issued by the Ministry of Transport (Notice No M 195 Revised Nov 1945) from which most of the following points are taken —

GENERAL PRECAUTIONS

- (1) Ships calling at malarious ports should lie as far off shore as possible
- (2) Communications between ship and shore should be curtailed or forbidden between dusk and dawn
- (3) A day or so before arriving in a malarious port all doors ventilators and portholes should be mosquito proofed Failing this mosquito nets should be supplied to all on board
- (4) No one should sleep on deck without a mosquito net
- (5) All possible lights should be blacked out
- (6) Everyone should be protected by suitable clothing after sun set Exposed parts (wrists ears etc) should be smeared with culifuges (dimethyl phthalate D M P —conveniently applied in a vanishing cream such as Mylol (Boots)
- (7) No bathing after dark
- (8) Anyone feeling off colour should report to the Medical Officer at once

MEDICAL PRECAUTIONS —Prophylactic drugs should be taken if the chances of infection are great A responsible officer should see that all persons receive and swallow the prescribed dose

Mepacrine should be given to adults in doses of one tablet

(0.1 gm) daily after food and followed by a copious draught of water. This suppressive treatment should be started ten days before the malarious port is reached and continued for thirty days after the risk of infection is passed. Smaller doses are necessary for children according to the usual formula $\frac{\text{Age}}{20} \times \text{adult dose}$.

Specific treatment—Depending on the drugs available any of the following three treatments may be used, the third being the one of choice.

1 Quinine gr 10 three times a day for seven days

Quinine sulphate (or hydrochloride)	gr 10
Citric acid	gr 30
Magnesium sulphate	gr 20 to 60
Spirit of aniseed	m 10
Simple syrup	fl oz $\frac{1}{2}$
Water	to 1 fl oz

2 Mepacrine 0.1 gm three times a day after food for five to seven days

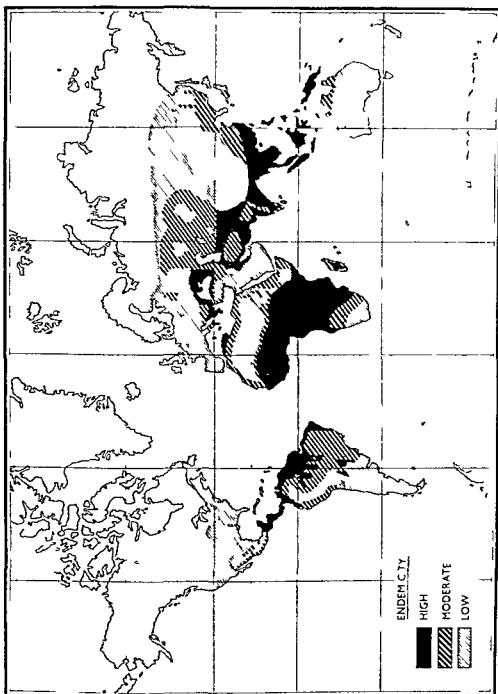
3 Quinine as in 1 for three days and then Mepacrine as in 2 for five to seven days

Treatment of cerebral and other forms of pernicious malaria is described on pages 34 et seq.

The four maps show in which part of the world some of the commoner tropical diseases can be expected to occur (Figs 33, 34, 35 and 36).

A NOTE ON WATER AND FILTH DISEASES—Sometimes the master may be obliged to take in water the source of which is suspect. It should then be chlorinated by adding one part of chloride of lime to 250 000 parts of water, i.e. 60 grains to 200 gallons. For successful chlorination 0.2 to 0.5 parts of *free chlorine* per million parts of water must be present. The concentration can be determined with the Chlorotex reagent (British Drug Houses) by mixing 50 c.c.m. of water with 5 c.c.m. of reagent in a cylinder and comparing the colour after one minute with that of a chart. Pink indicates 0.2 parts and red 0.5 parts per million. It may be necessary also to recommend chlorination in a typhoid or dysentery epidemic. Any objectionable taste may be removed by adding potassium permanganate (0.2 to 0.8 parts per million) before with or after treatment.

The criteria of good and bad food are given in the Ship



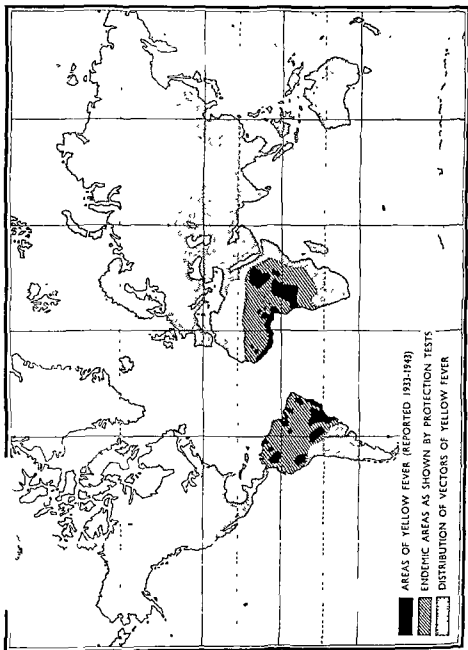
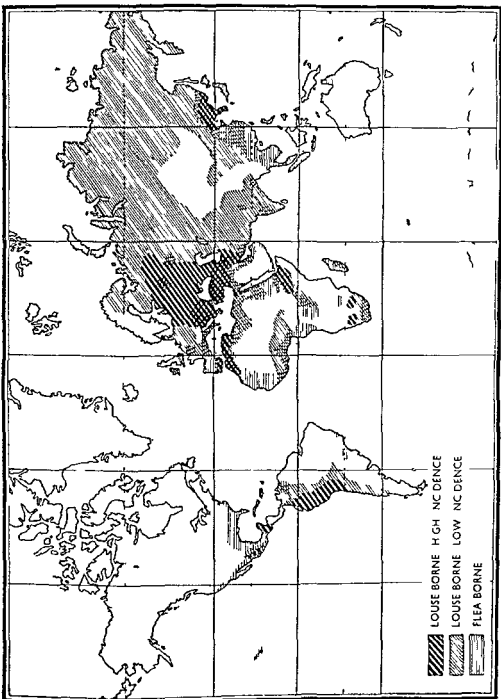
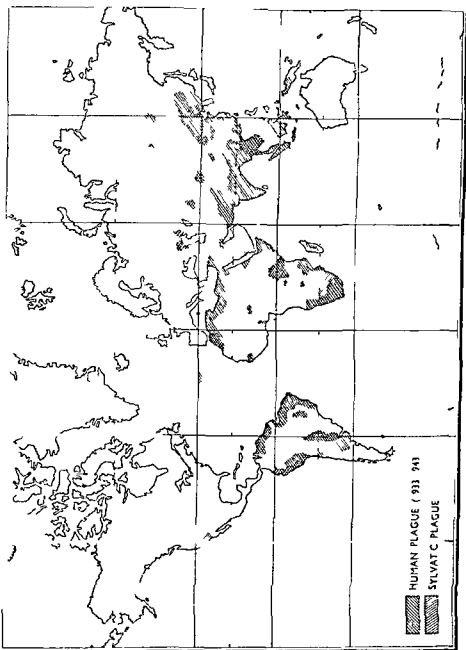


Fig 34. Yellow fever





Captain's Medical Guide A point however that needs to be stressed is the danger of buying fresh fruit and vegetables in foreign ports. They are very likely to carry dysenteric infections which are always endemic in the East during the warmer months. An order forbidding trade with fruit boats cannot be enforced and it is probably better simply to insist that the food stuffs should be immersed in weak potassium permanganate solution and then washed in running water if they are not to be cooked.

Stench

A compartment which has become foul smelling should be washed out with chloride of lime (bleach) and water (1 in 20). Metals must first be covered with grease to prevent corrosion. In war time corpses may have to be dealt with. Damp sawdust and bleach powder 5 to 1 will render the atmosphere tolerable. A similar situation might arise with decaying carcasses in peace time and the wearing of respirators affords protection while the cleaning is being effected.

POISONING ON BOARD SHIP

A variety of poisons more or less peculiar to ship life may cause emergencies at sea.

Alcohol

Acute alcoholism is common especially when a ship is in port and sailors may achieve an astonishing degree of coma *not infrequently associated with lead injury*. The condition is usually caused by the rapid consumption of an excessive amount of ordinary liquor but where doping is alleged methyl alcohol poisoning may have to be considered (*see page 10*). This should be suspected when dimness of vision and general symptoms come on after a latent interval of from nine to 36 hours.

The treatment of a severe case of alcoholism in coma should be by stomach washout (*page 391*) using warm sodium bicarbonate 60 grains to the pint where the patient is conscious vomiting should be encouraged and apomorphine hydrochloride subcutaneously gr $\frac{1}{10}$ is a convenient emetic.

Chronic alcoholism engenders difficulties of a different order. The doctor should keep aloof from alcoholics among the

passengers for although their company may be amusing, the association will not enhance his reputation

Ethylene glycol.

This substance used as a solvent for nitro cellulose and dopes ' has been taken as an intoxicant and caused many deaths It produces a hepato renal syndrome of which anuria is a special symptom Treatment is on the lines suggested on page 57

Carbon monoxide poisoning on board ship may occur alone or in conjunction with intoxication by nitrous fumes (*see page 4*)

Nitrous fumes result from incomplete combustion of cordite and are therefore likely to appear when a mine or torpedo strikes a ship carrying ammunition Symptoms and treatment are as described on page 315

Carbon tetra chloride may cause poisoning on board ship when used in fire fighting and minor symptoms have occurred when it has been used for cleaning clothes They are described on page 317

Methyl bromide is a special hazard in motor torpedo boats Its heavy vapour is most effective against fires involving high octane spirit In high concentrations, as in fire-fighting, pure gassing may occur but in the recent war nearly all cases resulted from accidental inhalation of much lower concentrations of the vapour For instance, slight leakage into the wardroom of a motor torpedo boat produced symptoms which were first interpreted as food poisoning A few hours later two of those affected went into convulsions and died while a third had staggering gait and other symptoms suggestive of alcoholic intoxication The illness remained obscure until somebody thought of the fire extinguishers and weighing of the cylinders showed that one was empty

There is no effective treatment but continuous oxygen and where fits are occurring sedation with Thiopentone B P (Pentothal) can be tried Paraldehyde 2 c cm in 20 c cm saline given intravenously is often valuable in controlling recurring convulsions In patients who recover, convalescence is often complicated by depression

Incidents of this kind emphasise the importance of thorough investigation whenever several men go sick with symptoms that are at all unusual

FISH STINGS

The effects of these and their immediate treatment are not as widely known as they should be. Many fish have sharp spines with a poison gland at the base. The lesions inflicted closely resemble snake bite, and occasionally tetanus bacilli are inoculated simultaneously. The initial pain, invariably described in the strongest possible terms, is accompanied by disabling prostration, which may explain some bathing fatalities otherwise unaccountable.

Treatment—This is on the same lines as for snake bite. The poison, a toxalbumin, is readily destroyed by potassium permanganate, a 5 per cent solution being injected into and along the track of the wound. (This solution has a deep port wine colour.) Failing this, the wound should be incised and crystals of potassium permanganate rubbed in. Relief of pain is instantaneous.

H. Muir Evans, in his book "Sting Fish and Sea Farer" gives this vivid account of a sting by a lesser weever which happened to his eight-year old son.

"While bathing a few yards from his home he was stung in the foot and brought home crying and in great pain. I happened to return to the house shortly afterwards and, having found the wound, immediately carried out the treatment as above described using a hypodermic needle and permanganate solution. The condition of the boy was alarming as he was very pale with a rapid feeble pulse and cold sweats. He seemed beside himself with pain and the insertion of the needle into the wound was not even noticed. Within a few minutes of the injection the pain was completely relieved, and after a glass of hot milk he was anxious to return to the beach. After an hour's rest he was allowed to rejoin his fellows, no swelling or inflammation followed the sting as its virulence was at once destroyed by chemical means."

UNDER-WATER HAZARDS

(1) *Caisson disease*.—A caisson is a device for enabling a number of men to work on the sea bottom or alongside wrecks, usually at a stretch of from six to eight hours, at a depth of 60

feet or less without the encumbrance of diving suits. Essentially it is a metal column at the bottom of which men work in a special chamber. Water is prevented from occupying the working chamber by the counter pressure of compressed air. Communication between the working chamber and the external atmosphere is effected by a series of airlocks in which men are gradually compressed or decompressed.

Caisson disease results when decompression is carried out too quickly when bubbles of nitrogen are formed particularly in those tissues with a poor blood supply (joints) or those containing much fat (spinal cord and subcutaneous tissues).

A similar condition occurs but more rarely in divers who work at greater depths but for shorter periods. The condition appears only when the pressure of the air is more than 18 lbs to the square inch i.e. below 40 feet of water.

SYMPTOMS —

- (a) Bends —liberation of bubbles into the ligaments of joints causing pain and flexion. These may not come on for five to six hours after reaching the surface.
- (b) Patchy discoloration of the skin.
- (c) Abdominal pain distension and vomiting.
- (d) Nervous symptoms—headache vertigo paraplegia coma and convulsions.
- (e) Deafness caused by low pressure in the middle ear.

Treatment —Caisson disease can be prevented by gradual decompression according to the Admiralty instructions. For divers a Davis Submerged Decompression Chamber containing oxygen is now often used. Thus the diver enters 60 feet below the surface (not deeper to avoid oxygen illness) and he can then be quickly raised to the parent vessel with much less risk of decompression sickness.

When symptoms are present it is necessary to re compress the patient to the original pressure at which he was working and then to decompress slowly over a period of about five hours. If no re compression chamber is available the emergency is best dealt with by placing the patient in a diving suit and lowering him to the bottom in the care of another diver.

Oxygen is also useful in bends and alkalies should be given as they increase the CO_2 combining power of the blood. Substitution of nitrogen by helium has also been tried but the

results are uncertain, nitrogen narcosis (caused by increased partial pressure of nitrogen) is however, avoided by this means in divers and men at work at great depths

(2) **Oxygen disease.**—Breathing of pure oxygen at a depth of 120 feet (four atmospheres pressure) produces acute oxygen poisoning because it interferes with the carriage of CO_2 from the tissues owing to lack of reduced hæmoglobin. Divers and submarine crews breathing pure oxygen while awaiting escape by the Davis Submerged Escape Apparatus are liable to be affected. Early symptoms are nausea and vertigo, twitching and loss of control of the lips and tingling of the extremities. Within an hour, convulsions, coma and death follow. Relief is obtained by breathing ordinary air, but in the case of the submarine crew awaiting escape this introduces a risk of Caisson disease. Men brought to the surface usually recover rapidly, convulsions ceasing and the patient falling into a deep stertorous sleep. Later there may be headache, vomiting and loss of memory, but no permanent injury.

Too rapid ascent of a diver breathing oxygen or a man in a Davis Submerged Escape Apparatus carries with it the risk of 'burst lung' due to trauma of the alveolar walls by the oxygen rushing out of solution. Hæmoptysis, not necessarily fatal, occurs.

Note.

(a) 'Burst lung' is not Caisson disease, since the subject is breathing pure oxygen. Workers in caissons and divers breathing *ordinary air* are *not* liable to acute oxygen sickness, since the partial pressure of oxygen necessary to produce symptoms would only obtain at a depth of 600 feet or more, which is outside the range of both types of worker.

(b) Chronic oxygen sickness (a pneumonia-like condition produced by breathing oxygen at a partial pressure of one atmosphere for many hours) could theoretically affect divers breathing compressed air at a depth of 150 feet. Under ordinary working conditions this does not happen since exposure is not long enough.

(3) Arsine and stibine poisoning

In the manufacture of submarine batteries scrupulous care is taken to exclude arsenic from the lead plates, but during the

last war there was at least one boatful of cases of poisoning. During charging batteries evolve hydrogen which contains traces of arsine if the lead or sulphuric acid contain arsenic. If the boat dives at the end of a rapid charge the batteries continue to evolve gas and the crew may be poisoned.

A similar accident may result from the formation of stibine from antimony present in the grid of the battery. Both arsine and stibine are essentially odourless and their toxicities are similar. Their presence can be recognised by the fact that silver nitrate test papers turn black.

Symptoms and treatment are essentially the same as in similar poisoning in industry (*page 319*)

(4) Immersion blast

Under water explosions produced this additional hazard to survivors from ships in war time. Ear drums may be ruptured and multiple hæmorrhages in the lungs cause dyspnœa and hæmoptysis. Perforation of or hæmorrhages into the gut may occur in several places either at the time of the injury or from six to 10 days later in the delayed cases an infected hæmatoma being present.

Treatment — Instruct sailors to swim on their backs as horizontally as possible if they are near a sinking ship when depth charges may explode. This will remove the air containing organs as far as possible from the explosive wave which becomes intensified at any air water boundary.

For the lung lesions absolute rest, morphine and oxygen are indicated. Where perforation is obvious laparotomy should be performed but nice judgment is required when the signs are less definite. If laparotomy is undertaken an inhalation anæsthetic should not be used owing to the serious hazards associated with blast lung.

MISCELLANEOUS CONDITIONS

The "acute abdomen" at sea

Should a perforated peptic ulcer be diagnosed and a surgeon not be at hand or the patient be judged to be unsuitable for surgery conservative treatment by gastric aspiration should be started.

After an injection of morphine the patient sucks a lozenge

of amethocaine (1 gram) and the stomach is then emptied by a large stomach tube and a Senoran's evacuator or syringe. This tube is then withdrawn and a smaller one of Ryle pattern introduced and aspiration continued at half hourly intervals for at least 48 hours. Parenteral chemotherapy should be started and glucose saline given rectally subcutaneously or intravenously. Sips of water may be taken but should be aspirated again at once. This treatment is more likely to be successful if adopted early and if leakage of gastric contents has been minimal.

Toothache and gum infections

Ships do not usually carry dentists and the sea going doctor will find it very useful to have some elementary knowledge about toothache of which there are three main causes —

(1) **Pulp infection** — This occurs secondarily to a cavity and the tissues surrounding the tooth are normal. Gentle probing will usually elicit which tooth is affected.

Treatment — Unless the infection is gross an attempt should be made to fill the cavity. Clean it out with an excavator after an injection of 20 to 40 minims of 2 per cent procaine round the tooth. Swab the cavity with oil of cloves. Fill the cavity with zinc oxide mixed with two or three drops of oil of cloves. This will relieve pain temporarily.

(2) **Periodontitis** — This occurs secondarily to pulpitis. The tooth is tender on pressure raised in its socket and pus may appear round it.

Treatment — Give very hot mouth washes of Chloro-cresol B.P. (Dettol) 12 drops in a tumbler of water. The liquid should be held by the tongue around the tooth. Never apply hot fomentations to the cheek. Extraction is often necessary when pus is present and should always be done under general anæsthetic injections into infected tissues being dangerous.

(3) **Infection round erupting lower wisdom teeth** — The patient complains of a dull pain sometimes associated with trismus. There may be an inflamed gum flap over the tooth. Mouth washes should be given and the flap painted with tincture of iodine.

Ulcerative gingivitis (Vincent's angina)

This is characterised by sore and bleeding gums and sometimes tonsillitis is present also

Treatment—In the acute phase stop brushing the teeth and discard the old toothbrush. Clean the teeth with cotton wool and wash the mouth out with Dettol or hydrogen peroxide thrice daily. Apply 10 per cent chromic acid carefully to the gums around the teeth on pledgets of cotton wool. Follow this with hydrogen peroxide similarly applied. Black chromic oxide is immediately produced and should be allowed to remain in the tissues for about a minute following which a further hydrogen peroxide mouth wash should be given. These pain relieving measures can be usefully supplemented by penicillin either systemically or locally for three or four days.

Note

If the master or a senior ship's officer gets Vincent's angina don't treat him with organic arsenical preparations. It will be round the ship in half an hour that the Old Man is on Nabs with considerable detriment to discipline.

External otitis

This condition is predisposed to by wax in the ears and by tropical climate which produces a sodden macerated meatal skin. It results more frequently from infection from a dirty towel than from bathing. Those who wear head phones are particularly liable to suffer.

Treatment In the tropics wireless telegraphy personnel should have their ears inspected every week their external meatuses cleared of wax and 70 per cent spirit drops instilled. Bathers should be instructed to drain the water out of the ears by tilting the head to one side and in those predisposed to infection vaselined cotton wool can be inserted before bathing. Other people's towels should not be used.

When the condition is established gently syringe the meatus with 2 per cent bicarbonate solution to remove debris and dry carefully with a cotton wool swab. Then apply drops of 10 per cent mild silver proteinate B.P.C. (Argyrol) in spirit. For the more acute case with constitutional symptoms pack the ear with gauze wick moistened with 8 per cent aluminium acetate solution keeping it moist from a drop bottle for 24 hours after which

it should be changed Hydrogen peroxide and fomentations should not be used

THE MANAGEMENT OF PSYCHIATRIC CASES

The treatment of anxiety states and hysteria is much the same at sea as on land, but where dangerous psychotics are concerned special circumstances obtain. Thus, the means of suicide and manslaughter are ready at hand, panic is easily engendered and vitally important machinery may be tampered with.

In handling cases of acute psychosis the ship's doctor should bear in mind the following points —

(1) Acute mania is sometimes the presenting symptom of heat-stroke (*see page 305*) and severe cerebro-spinal meningitis, but if these two diseases are thought of, mistakes are unlikely to be made.

(2) Having decided that the patient is undoubtedly mad, the exact diagnosis does not matter very much. The problem is a short term one, namely, how best to get the patient to a port in a reasonable state of nourishment or without having done himself or others harm. Any form of treatment which will bring this about is justifiable, but when forcible restraint is used, *medico-legal points arise (see page 346)*.

(3) The most dangerous period as regards suicide is in the *recovery* stage of acute depression. Most suicides are surprises. It is not true, however, that those who talk of suicide never commit it.

Treatment — Put the patient in a spare cabin devoid of removable fittings, search him thoroughly, and have him watched night and day. See that he gets adequate nourishment, even if this involves forced feeding. To control extreme violence, use a strait jacket, a Neil Robertson Stretcher (Fig. 37), or roll the patient in a sheet secured with safety pins.

In severe manic cases, hyoscine hydrobromide gr $\frac{1}{100}$ and morphine sulphate gr $\frac{1}{2}$ subcutaneously are often necessary. Sedation can be kept up with oral paraldehyde 120 m. or sodium amytal gr 3 in capsule. In milder cases, nembutal gr $1\frac{1}{2}$ to 3 or sodium barbitone (medinal) gr 10 are useful.

MEDICO-LEGAL PROBLEMS AT SEA

It is difficult to deal briefly with these, and in many cases

definite rulings have not been laid down. Certain basic principles however, are stated below much help in compiling these having been obtained from A. V. Elder's 'Ship Surgeon's Handbook'.

(1) Status of the doctor.

The word of the master is law and the doctor must therefore obey his orders. If there is disagreement the doctor must state

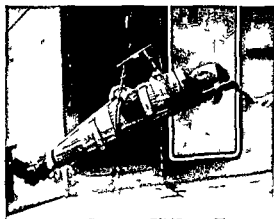


FIG. 37

The Neil Robertson stretcher

his case in writing and the legality or otherwise can be established later in harbour the matter being laid before the owners' agents or British Consul. If for example there were differences of opinion as to when a sick man was fit to resume duty the master could have the final word though it is extremely unlikely that he would interfere where a medical subject was in question. Again the point might arise while embarking third class passengers at the gangway. Thus Elder rejected a case of advanced locomotor ataxia with incompetence of the sphincters while the agents urged acceptance on the plea of loss of passage money. In a case of this kind the master might have the last word and could overrule the doctor though again he would be extremely unlikely to do so.

(2) Fees

The legal position is that the doctor must treat members of

the crew and steerage or emigrant passengers, gratis. Some lines extend this service to saloon or second class passengers, but allow acceptance of any fees offered. Others attempt to limit fee-taking to illnesses which did not originate on the voyage. In practice this is very difficult to decide, and in any case if the patient will not pay there is no effective machinery to make him. As Elder says the doctor has no time to bother about proceedings for recovery, and would probably be looking out for another ship if he did. Bad debts will inevitably be incurred at sea as on land.

(3) Conflicting duties.

In the ship the doctor has three loyalties —

(a) To the ship's company as a whole. Here he is acting in the capacity of Medical Officer of Health, with right of entry into all cabins and authority to advise detention of the ship in quarantine.

(b) To the shipowners and to the Board of Trade. They will require records of sickness and work done, at any rate in respect of third class passengers and crew.

(c) To his patients, in respect of professional secrecy. The following incident illustrates the above points (Elder) —

'A North Atlantic surgeon whose ship was laid up, took a post as locum tenens in the Midlands. During his time there he saw and treated a patient for epilepsy. Some six months later, while examining passengers on embarkation he recognised his late patient among them. Epilepsy being on the prohibited list, his position was awkward, to say the least of it. He allowed the passenger to embark, watched him during the voyage across, and as no fit occurred he said nothing. The sequel took place a few months later. As a result of an epileptic seizure the victim was taken to the immigration station at Ellis Island where he admitted his complaint and added that the surgeon in the ship in which he had crossed was quite aware of it, having treated him for it previously. The immigration authorities took the matter up with the steamship company and the surgeon. His attitude was, that as his knowledge of the patient's complaint was gained quite apart from his employment in the steamship company's service, or on the American route, he therefore considered it as 'privileged' and no part of his duty to report

it officially Whereupon the patient was deported and the matter dropped

(4) *Forcible restraint of lunatics*

If possible obtain a second and independent certificate inform the master and make an official entry in the log book If the patient is landed the reason for this should be clearly stated on the certificate as normally it is an offence to land a passenger at a wrong port without his consent

(5) *Accidents*

Full and careful records should be kept of all accidents as litigation is often a sequel If the surgeon has to continue treatment for an accident sustained before embarkation a statement will be asked for at the end of the voyage Elder recommends a certificate stating that the holder has been treated between certain dates and nothing more This will avoid discrepancies

(6) *Alcoholism*

Drunkenness is an offence under the Merchant Shipping Act and punishment can be inflicted by the master The doctor will be called upon to state whether a man is drunk or not and usually a definite opinion has to be given— drunk or sober no intermediate condition being recognised

Chronic alcoholism is a more difficult problem as it is not a crime and the doctor can take no definite action unless the offender becomes a patient when temporary abstinence can be enforced (Incidentally when a responsible officer drinks excessively putting him sick is often the best course) If a chronic alcoholic becomes a public nuisance and passengers complain it is the master and not the doctor who must take action *Although it will appear from this that pitfalls in the doctor's path are many yet in practice they are usually avoided by a little tact and common sense*

C ASTLEY CLARKE

CHAPTER XXI

Medical Emergencies in the Air

MEDICAL emergencies in the air arise essentially as a result of the environmental conditions of flying. In this chapter will be considered both those emergencies which are psychological or physiological in origin, being reactions of the healthy subject's mind or body to the abnormal environment, and those which are pathological, the exacerbation or complication of an existing lesion by such environment. Examples of the former are anoxia and decompression sickness, of the latter, the effect of altitude upon a pneumothorax, or perforation of a peptic ulcer.

EMERGENCIES DUE TO HIGH ALTITUDE

These may be caused by decompression, anoxia, and cold.

Decompression.

The symptoms of decompression sickness are not normally encountered even in susceptible people below 30,000 feet although abdominal discomfort caused by expansion of gas in the colon may occur from 20,000 feet upwards. After thirty minutes or more at heights of 30,000 feet or above, various alarming symptoms leading to collapse and, in rare cases, to death may occur. They result from the release of bubbles of nitrogen into the circulation, body fat, central and peripheral nervous system and tissues generally.

The most notorious are the "bends," acute pains in the muscles and joints, especially in the shoulders, elbows and knees. Rubbing or exercise will occasionally abate the pangs, and local pressure with a sphygmomanometer cuff has abolished peripheral skin and muscle symptoms. Pain may become so severe that acute shock with pallor, sweating and collapse is precipitated if descent is not made. Severe abdominal pain is also encountered. Once the emergency has occurred, descent to restore the pressure is imperative.

More serious, because more dangerous to life and more liable to leave enduring effects after descent, are symptoms arising in the central nervous and respiratory systems. Such symptoms may

be restricted to an urticarial reaction in the skin area supplied by the affected nerve but severe pain along the whole course of a peripheral nerve is not uncommon. Cases of paraplegia, epileptiform convulsions and coma have been described. In other subjects collapse of central origin may appear almost unheralded by other symptoms. The patient suddenly becomes intensely pale obviously in urgent distress with slow or practically absent pulse before losing consciousness completely. Convulsions and visual scotomata may persist after return to ground level.

In the chest symptoms tend to be particularly distressing and may induce a state of violent apprehension and anguish reminiscent of angina pectoris. Retrosternal pain, often violent and stabbing or a generalised burning sensation in the lungs, announce the obstruction to the pulmonary circulation by minute bubbles of nitrogen. Oedema of the lungs rapidly follows leading to paroxysms of coughing which are unproductive as long as altitude is maintained because the rarefied air will not support the expulsive efforts of the respiratory muscles. If this situation is not relieved death may follow within a few minutes or may occur later after descent from cardiac failure resulting from pulmonary oedema and massive hydrothorax.

DIAGNOSIS—Above 30 000 feet the onset of any of the symptoms described above is virtually diagnostic.

PREVENTION—Escape of dissolved nitrogen can be prevented by—

- (1) Restriction of height
- (2) The use of pressurised cabins
- (3) Sufficiently gradual ascent
- (4) Elimination of nitrogen by prolonged breathing of pure oxygen prior to ascent at pressures not greater than one atmosphere

Treatment—The most pressing indication is descent preferably to as low an altitude as the pilot can sanction but at least below 25 000 feet. In patients with collapse, convulsions or pulmonary symptoms the flight should be abandoned. In all cases the inhalation of pure oxygen should be maintained for as long as any symptoms persist.

Treatment for shock by rest and warmth may have to be continued after landing. Continued observation, oxygen and venesection may be needed in cases of pulmonary oedema when

right sided heart failure threatens. Despite the alarming nature of these symptoms, the prognosis following recompression is almost always good. The probability of recurrence on subsequent ascents must never be forgotten.

SPECIAL EMERGENCIES—Certain pathological conditions will be particularly exacerbated by ascent to high altitudes. These



FIG 38

Right sided pneumothorax at ground level

include all cases of pulmonary disease treated by artificial pneumothorax, diaphragmatic herniæ and chronic infection of the sinuses or middle ear.

Owing to the comparative increase in the pressure exerted by contained gas in the abdominal viscera at high altitude perforation of a gastric or duodenal ulcer is even more catastrophic than at ground level, owing to the greater tendency of the visceral contents to escape into the peritoneal cavity. The effect upon an artificial pneumothorax and on the colon is illustrated in Figs 38, 39 and 40.

Treatment—The outstanding and immediate indication is a return to a lower altitude

Acute pain caused by changes in atmospheric pressure

In contradistinction to the effects of decreased atmospheric pressure acute pain in the sinuses and middle ear and in imperfectly filled teeth may be produced by increased pressure on swift



FIG. 39

Same patient as in Fig. 38 at altitude of 9 000 feet showing increased collapse of right lung and mediastinal displacement

descent. Such pain can be considered an emergency only when it produces unconsciousness from bradycardia and falling blood pressure.

During ascent escape of air from the Eustachian tubes and ostia of the sinuses is gradual and automatic and only a transient sense of fullness results. The reverse process is less spontaneous and may be arrested if catarrhal inflammation is present. The

resulting pressure differences give rise to acute pain. These effects may occur as low as 15 000 feet.

Dental pain is rare below 25 000 feet in common with decompression sickness. It disappears at lower levels but the pain of sinus and otitic baro trauma persists.



FIG. 40

Normal subject after barium meal at 35 000 feet. Striking distension of the cæcum and ascending colon.

Treatment—The first essential in these cases is a controlled and gradual descent. Auto-inflation of the Eustachian tubes by holding the nose and blowing may prevent symptoms if begun early in the descent. Decongestive nasal drops or a benzedrine inhaler may also be used prophylactically. No patient with Eustachian catarrh should be permitted to fly above 5 000 feet.

Anoxia (altitude sickness).

Although anoxia is the most notorious of the physiological problems of flight, it is important to realise that the physical mechanism of its production is simply one aspect of the generalised fall in pressure with increasing altitude.

Anoxia in flying is caused by the decreased partial pressure of oxygen in the rarefied atmosphere. It constitutes a far greater hazard at all altitudes above 10,000 feet, with eventually greater danger to life, than any other physiological emergency. Its effects become significant at considerably lower altitudes than those of decompression sickness.

For this reason the use of additional oxygen for all flights of more than an hour's duration at 10,000 feet or of whatever duration above 15,000 feet is compulsory in the Royal Air Force. This is accomplished by the inhalation of oxygen in increasing proportions with air through special masks from apparatus designed to cover the subject's requirements up to 37,000 feet. Above this height even pure oxygen breathed continuously is not sufficient to prevent anoxia, as the total atmospheric pressure is less than the partial pressure of oxygen needed to saturate the blood at ground level. Above this height some form of additional pressure is necessary to maintain the normal oxygen exchange in the lungs, and to secure this without damaging the alveoli, it is normally necessary to encase the subject in a pressure suit or cabin.

For civilian passengers under peace time conditions, the use of oxygen masks is not commercially advisable. Such passengers are neither trained, selected, nor even necessarily fit for such dependence upon the wearing of special equipment for their safety. The impossibility of permitting smoking under these conditions is, moreover, a serious commercial disadvantage. At most oxygen masks and apparatus in passenger aircraft are likely in the future to be provided solely for temporary use in emergency. The problem of anoxia will therefore be met either by restricting the height of flights even further than was foreshadowed in the section on decompression sickness or by providing pressure cabins.

SYMPTOMS AND SIGNS—At heights below 20,000 feet or when the rate of production of anoxia is slow, the effects are at first insidious. The patient's judgment, powers of self criticism, and attention suffer, while subjectively he may experience either

euphoria irritability apathy or depression Aggressive tendencies are not uncommon Performance of all kinds is rapidly impaired and inaccuracy is characteristic At this stage the most valuable objective sign is cyanosis particularly of the lips ears and nail beds

Later diminished hearing and visual acuity become marked jactitations appear there is gross impairment of fine movements and muscular power is lost progressively By now the patient is aware of his plight but not of its extreme danger His respirations become alternately quicker and slower than normal tachycardia develops jactitations increase in frequency and severity until extremely cyanosed helpless and drowsy he becomes comatose develops convulsions and eventually dies

At heights over 25 000 feet if oxygen deprivation is sudden the patient rapidly loses consciousness lies twitching and sweating and may die in coma within an hour if untreated Another extremely important factor is the degree of cold to which the patient is exposed

Diagnosis—The nature of the symptoms and the manner of their appearance should suggest the diagnosis in all cases where the possibility of anoxia exists The most significant individual sign apart from the patient's behaviour is his marked cyanosis

Treatment—The emergency usually arises only through failure of the oxygen supply Its restoration is the most urgent indication in treatment In early or mild cases this is all that is required All symptoms disappear and there is no need for the aircraft to descend Since judgment and insight are lost early in anoxia the decision to use oxygen must devolve on someone other than the patient

If complete recovery is not immediately secured as after very severe exposure descent is advisable If respiratory paralysis is present artificial respiration with inhalation of a mixture of 93 per cent oxygen and 7 per cent carbon dioxide should be used

The relationship between cold and anoxia dictates that treatment should be accompanied by all possible measures to maintain the patient's body heat by cabin heating if possible warm blankets and hot drinks

Following landing the excessive fatigue which often follows anoxia is relieved simply by rest in bed Such patients may sleep for 24 hours almost continuously

SPECIAL CONDITIONS—Patients suffering from any degree of cardiac failure or decompensation or from pulmonary disease will naturally be more susceptible to anoxia develop symptoms at lower altitudes and be exposed to considerably greater danger from such circumstances than healthy passengers. Air travel is generally contra indicated for such patients although if for any



FIG. 41
Frostbite

reason it is imperative the margin of safety can be increased by limiting the altitude flown as far as is practicable and by placing them in the care of a competent attendant who will ensure that they receive sufficient oxygen to forestall the slightest degree of cyanosis.

EMERGENCIES DUE TO COLD

With ascent the temperature falls at a rate of approximately 2°C for every 1 000 feet above sea level. This continues until a temperature of minus 55°C is reached at which point the temperature in the sub stratosphere remains roughly constant. The intense cold produces frostbite and also general effects

Frostbite.

This only occurs when the air temperature is well below zero. The nose, ears and hands are common sites (Fig. 41) and an early sign is a hard white patch on the skin detected in making a face. At minus 35°C contact of the skin with a metal surface produces instantaneous frostbite of a severity comparable with that of a second degree burn of similar extent.

PREVENTION—Adequate protective clothing should always be provided and the wearing of gloves strictly enforced. Beards and moustaches should be kept short. In the air prevention depends on efficient cabin heating or in the case of combatant air crew exposed in unheated turrets upon electrically heated clothing. These measures must be combined with adequate oxygen administration. The skin of the face can be protected by lanoline which lowers its freezing point. Studs and metal fittings must not come into contact with the skin.

Treatment—While in the air all that can be done is to rest the affected part and administer oxygen and hot drinks. Following landing treatment should be continued along similar lines the part being protected if necessary by cotton wool and kept at cool room temperature. No attempt should be made to hasten recovery of local warmth and sensation.

An alternative method involving immersing the part in cold water which is very gradually warmed and re-cooled if pain increases has been recommended but under no circumstances should massage or rubbing with snow be employed. In all cases where lividity and induration persist transference to hospital is indicated.

GENERAL EFFECTS—Intense cold apart from its disagreeable sensation and depressing and demoralising influence predisposes to tissue anoxæmia and a lowered oxygen tension in the blood. A vicious circle is created in which extremely low temperatures predispose to anoxia while among the effects of anoxia is a diminished resistance to cold. It is therefore exceedingly important that both body temperature and oxygenation be safeguarded during flight at high altitude and that in calculating the quantity of additional oxygen required for full efficiency at any altitude allowance should be made for the proportionately greater oxygen debt created by cold.

For civilian passengers the only safe answer to these problems

is a heated pressurised cabin. Emergencies due to decompression anoxia or cold can then only arise through a failure of the pressure and heat. This must be met by immediate descent below 20 000 feet and the responsibility for this rests with the pilot.

It rarely happens that passenger carrying aircraft have cause to fly at or above 30 000 feet but neither are they likely to limit their altitude to below 10 000 feet which is the practical ceiling if the physiological problems so far described are to be simply evaded instead of overcome.

Transport aircraft must be able to fly over bad weather instead of through it if schedules are to be maintained they must be able to rise above turbulent air to spare their passengers motion sickness and on long trips they operate far more economically in the rarefied atmosphere at comparatively high altitudes. All this is possible for them without the least risk to their passengers if they are fitted with air conditioned adequately heated pressurised cabins. A differential pressure of five or six pounds to the square inch is possible and satisfactory. This enables an internal pressure equal to that at sea level to be maintained at all heights of the aircraft up to 11 000 feet.

The supreme potential emergency inherent in this state of affairs is a failure of the pressure cabin. If this is partial or gradual and is detected by the pilot all untoward effects can be averted by a smooth descent timed to compensate for the leak. If undetected mechanically the gradual and insidious onset of symptoms appropriate to the height flown occurs among passengers and crew as already described and reveals the potential danger.

There remains the dramatic possibility remote in peace time but ever present in war of the sudden and complete failure of the cabin (Explosive Decompression). Taking a pressure differential of five pounds to the square inch as standard it has been proved experimentally that the maximum rate at which decompression can occur even if one complete end of the cabin is blown off is equivalent to an ascent of 160 000 feet per second. Further experiments have demonstrated that decompression at this rate was well tolerated no more than discomfort being produced in most cases by abdominal distension while colicky pains even when severe were never incapacitating. The Eustachian tubes were blown open at once and remained open without discomfort until equilibrium was restored. The effects of explosive decompression

are in fact determined by the ultimate altitude attained. Thus below 30 000 feet decompression sickness is not a factor no matter how rapid and complete the cabin failure while the acuteness of onset of anoxia will depend entirely upon the actual height of the aircraft at the time.

For the utmost possible safety in any aircraft flying above oxygen height even if there is a pressure cabin the crew should be equipped with an emergency oxygen apparatus complete with individual masks. Below 30 000 feet even in the event of explosive decompression they would have ample time to put on their masks and bring the aircraft down to a safer altitude.

An extension of this principle to the provision of similar equipment for every passenger would be a wise precaution analogous to the provision of lifebelts on board ship. It is probable that the occasions for the emergency use of either would be equally infrequent.

LOSS OR IMPAIRMENT OF CONSCIOUSNESS IN THE AIR FROM OTHER CAUSES

Apart altogether from the effects of altitude there remain a number of other factors of varying importance which may lead to impairment or loss of consciousness while flying. They may be considered under the following heads but several distinct causes may be acting in any particular case.

- (1) Effects of acceleration
- (2) Asphyxia from carbon monoxide poisoning
- (3) Psychogenic disorders
- (4) Epileptic attacks
- (5) Cardiovascular instability syncopal or vasovagal attacks
- (6) Vertigo of aural origin
- (7) Miscellaneous disorders e.g. cramps caused by sweating or hyperpnœa

Emergencies due to acceleration

Speed by itself is in no way harmful to the human body provided that protection is afforded from wind but changes in rate or direction of motion of sufficiently high order can impose very great mechanical stresses upon the occupant of modern high speed aircraft.

Such accelerations or decelerations are of two kinds those associated with catapult or rocket assisted take off or with sudden arrest in crash landing or forced descent into water by a land plane and those involved in aerial manoeuvres at high speed such as tight turns or pulling out from a fast steep dive. Both types of problem are important in service aviation but neither has at present any direct bearing upon passenger flying except that the possibility of a forced landing can never be entirely excluded.

The unit of acceleration is referred to as G and corresponds to the acceleration due to gravity giving a body its normal weight and equal to a rate of fall in a vacuum of 32 feet/sec /sec.

Even if injury from hard projections inside an aircraft can be avoided the violent deceleration of a crash landing which may be of the order of $25\ G$ or more may cause internal damage to the body unless it is adequately supported. The implication of this is that emergency crash positions should be allocated to all passengers so that their bodies can be supported over as wide an area as possible in the direction of motion at the moment of deceleration by some comparatively unyielding part of the aircraft.

In tight turns or pulling out from dives the value of G produced by centrifugal force is considerably less and is acting normally in a physiological direction namely vertically downwards in a seated man but if raised over $5\ G$ or acting for more than five seconds it is liable to produce symptoms attributable mainly to disturbance of the circulation. At $7\ G$ the blood has an effective weight equal to that of iron and a blood pressure of over 200 mm Hg in the ascending aorta is necessary to maintain the cerebral circulation. Similarly venous return will be hampered by the tendency of the blood to pool in the legs and abdominal viscera and cardiac output will diminish as a result.

SYMPTOMS —The earliest sensation is that of increased weight of the whole body especially of the head and limbs. This is particularly noticeable if the subject attempts to move and at $4\ G$ or $5\ G$ he will be unable to lift his feet from the floor. It is followed by a progressive failure of vision known as blacking out from failure of the retinal circulation which

because of intra ocular pressure, precedes failure of cerebral circulation. Further increase of 'G' produces unconsciousness with flaccidity after blackout is complete, but never before. Diminution of "G" permits immediate recovery, though after sufficiently severe or prolonged exposure to 'G' disorientation and retrograde amnesia may persist.

DIAGNOSIS—Subjectively, this may be self evident. "Blacking out" rarely occurs with values of 'G' less than 3. If it does, vasovagal attacks or petit mal must be considered.

Treatment—Protection of the circulation against centrifugal force can be achieved in some measure by voluntary increase in muscular tone, by appropriate posture designed to diminish the length of the blood columns above and below the heart and by the provision of special garments to support and reinforce venous return from the legs and abdomen. These measures have no application to civilian passengers in transport aircraft.

Centrifugal force acting in the reverse direction from heart to head as in an outside loop or inverted spin is entirely unphysiological and exceedingly dangerous as reinforcement of the normal carotid blood pressure by increased "G" leads rapidly to retinal and cerebral hæmorrhages probably ending fatally. *It is rigorously avoided in service flying and no possibility of it should arise in civilian aircraft.*

Asphyxia from carbon monoxide.

This is only likely to be seen in obsolete poorly designed and maintained aircraft. (*For treatment see page 4*)

Loss or impairment of consciousness of psychogenic origin.

The most frequent cause of neurosis connected with flying is fear. This is a natural reaction to its extreme hazards under wartime conditions but is offset to a varying extent in service aircrew by their excellent training, and personal qualities.

For civilian passengers the dangers of flight are minimal but their mental approach is entirely different such confidence as they possess being based upon ignorance of the remaining dangers coupled with salutary confidence in the aircraft and its crew. The intrinsic insecurity of being airborne however, may induce a state of emotional tension in those predisposed to neurosis. Any alarming occurrence in the air such as violent

rocking or bumping as in bad weather or the appearance of ice on the wings may produce panic or hysterical confusion.

Awareness of height is not normally a factor of importance during the first 10 or 15 hours of flying but it tends to appear later sometimes with profoundly disagreeable intensity in those in whom a fear of height has always existed.

The mechanism by which acute anxiety during flight leads to impairment of consciousness, hysteria or confusion with subsequent amnesia varies in different individuals but acts essentially —

- (1) By producing an hysterical state of stupor or amnesia
- (2) By causing panic during which the subject may lose all self control and appreciation of immediate events
- (3) By causing hyperventilation at altitude which may result in anoxia and acapnoea
- (4) By producing secondary cardiovascular inefficiency which may cause syncope or blackout at low G

DIAGNOSIS —Hysteria or stupor in the air differ in no essential from similar manifestations on the ground. In service practice the diagnosis of hysteria has been reserved by definition for cases in which mental or physical symptoms not of organic origin are produced and maintained by motives never fully conscious directed at some real or fancied gain to be derived from such symptoms. This gain may be an honourable release from flying duties or among civilians the cessation of a particular flight. Differential diagnosis from other forms of unconsciousness such as epilepsy or syncope remains a clinical problem unaffected by altitude but the additional possibilities of anoxia or decompression effects have to be excluded in the air if environmental conditions are compatible with them.

Treatment —This consists simply in firm encouragement and reassurance. The infectivity of fear among passengers unused to flying suggests that attention should also be devoted to the prevention of anxiety among the other occupants of the aircraft.

Panic should be forestalled by encouragement and example whenever possible. Once it has developed prompt and decisive measures including if necessary physical restraint may be required to prevent the subject from harming himself or others or even trying desperately to escape from the plane.

People with a history of claustrophobia or a morbid fear

of heights are totally unsuited to travel by air, and should never be encouraged or persuaded to do so under normal conditions.

Hyperventilation among civilian aviators has been reported and is considered to be due to *emotional disturbances* when it cannot be accounted for by anoxia, physiological or pathological abnormalities. That fear and anxiety produce, among their other somatic manifestations, marked changes in the respiratory rate has been known for many years. Anoxia, and in susceptible patients, epilepsy, may result.

Epilepsy in the air.

Epileptic attacks appear to be relatively more frequent in the air than on the ground, suggesting the existence of some special precipitating factors due to flying. Cases fall into two groups: those with a history of attacks on the ground, and those where the first and perhaps only attack occurs while flying.

The precipitating factor in the second group may be physical (e.g. anoxia) or psychological. Men in this group might, but for flying, never have had a fit at all. The possibility of precipitation of an attack by the physiological stresses of flying is theoretical. Fear may cause reflex epilepsy just as may anger or any other stimulus. A fall of blood CO_2 may precipitate an attack in an epileptic, unconscious overbreathing from fear may act in this way.

Treatment—There are no special measures to be adopted in the air other than giving oxygen during the stage of cyanosis.

Cardio-vascular instability—syncopal attacks.

Fainting in the air in the absence of anoxia is uncommon except in predisposed persons. A lowered threshold to the effects of "G" together with a liability to faint in the air may be seen in patients whose vascular tone has been diminished by neurosis.

Among aircrew this is of course exceedingly dangerous and constitutes, while it persists, an absolute bar to flying duties. In passengers it raises no problems specific to their environment other than that of adequate oxygenation.

Vertigo.

Spatial disorientation in the air, especially when flying through cloud or during aerobatics, is natural among the inexperienced. *Aural vertigo of pathological origin may, however, lead to*

confusion amounting in some cases to impairment of consciousness. This again is only of dire significance among the aircrew but may be unpleasant for a passenger.

The condition is unrelated to altitude and its diagnosis rests upon the characteristic pattern of the attack. This is a sensation of falling and turning usually in one constant direction and often accompanied by profound nausea. It may provoke air sickness.

Treatment—No immediate treatment is necessary other than laying the patient down in the most comfortable position possible.

MISCELLANEOUS DISORDERS

Violent cramps due to salt loss through sweating occurred in one case seen by the writer. The patient was a muscular pilot who had always been subject to hyperidrosis. He was completely cured by the administration of salted beef extract drinks before and during flying. Tetany following hyperpnœa has been reported but is extremely uncommon.

In all cases where an emergency occurs in the air it is the responsibility of the physician to bear in mind all the special factors created by the environment but to realise that these are limited and that in modern passenger flying their effect should be minimal. Failure to recognise anoxia and decompression sickness would be regrettable but obsessed by such possibilities to overlook diabetic coma or a subarachnoid hæmorrhage would be equally unfortunate.

Air sickness

Air sickness deserves consideration here only in its most severe form that of a violent and unrelenting catastrophe which leaves the victim utterly prostrate sick unto death and almost desiring it completely unfit for any form of physical or mental activity.

Happily like all other forms of motion sickness air sickness is never fatal and improvement always begins from the moment of landing although in the more susceptible up to 24 hours rest may be required before the patient feels normal again.

Symptoms—The onset with nausea yawning pallor restlessness and apprehension often accompanied by headache and a desire for fresh air is followed sooner or later by vomiting which tends to recur at shortening intervals until the stomach is emptied when retching extreme malaise and profound misery

and despair encompass the sufferer. The subjective sensations can probably never be entirely appreciated by those who have not endured them. The victim's absolute incapacity for effort of any kind is a striking feature of the most severe cases.

DIAGNOSIS—This is not usually difficult. In milder cases distinction has always to be made between true motion sickness and the visceral accompaniments of neurosis. In the violent forms however this distinction is purely academic at least while the flight lasts.

Treatment—The accepted prophylaxis of a light digestible carbohydrate meal devoid of fried or fatty foods followed by a mild sedative such as Chlorbutol gr 15 (Chloretone) or a more specific prescription—

Hyoscine hydrobromide	gr $\frac{1}{100}$
Phenobarbitone	gr $\frac{1}{2}$
Benzedrine sulphate	gr $\frac{1}{4}$

is absolutely sound. This is far more likely to succeed if accompanied by a cheerful and confident approach to the flight by the patient not always easy to secure if he has suffered before. During actual attacks in the air little can be done except to ensure that the patient is receiving ample oxygen, is completely warm and lies preferably on his side as comfortably as he can. Cold and anoxia predispose to air sickness in susceptible people. Strychnine gr $\frac{1}{20}$ intramuscularly has been suggested in view of its effect in decreasing the vestibular reaction. In practice it is extremely doubtful if the discomfort of its administration can be justified by any observed effect. In aircrew receiving their oxygen through masks air sickness can be complicated by severe anoxia as the patient has continually to remove his mask to vomit. In such circumstances immediate abandonment of the flight is indicated. Following return to land rest warmth and hot sweet tea or other suitable drink will usually restore the patient rapidly.

SPECIAL CONDITIONS—Patients who through old age debility cachexia or cardiac disease cannot safely endure the prolonged vomiting tachycardia and initially raised but subsequently lowered blood pressure caused by air sickness should not be exposed to the risk of it at all. The tendency to motion sickness of any kind can usually be discovered from a careful history.

D STAFFORD CLARK

CHAPTER XXII

Medico-legal and other Non-clinical Emergencies

ALMOST all forms of treatment and investigation we use may on occasion be harmful to the patient and so lead to legal action. Every practitioner should therefore belong to a defence society (preferably the same one as his partner or assistant) and report to it *at once* when involved in a situation likely to lead to a claim for damages. At the same time immediate and energetic steps should be taken to reverse the consequences of the accident. Special medico-legal emergencies arising on board ship are described on *page 344* and the question of responsibility for advice given by wireless in an emergency is dealt with on *page 323*.

NEGLIGENCE

Since a charge of negligence may follow the doctor's attention to his patient a clear statement of what negligence means is desirable. In law negligence is judged by the standard of prudence of an ordinary reasonable man but a person who undertakes something requiring special knowledge or skill is negligent if by reason of his not possessing that knowledge or skill he bungles although he does his best. The negligence does not consist in the lack of skill but in undertaking the work without skill. The physician must exercise such care as a normally skilful member of his profession may reasonably be expected to exercise. This principle applies even though the doctor undertakes to do the work gratuitously. Extraordinary skill however is not required of any one and erroneous judgment in a difficult case does not constitute negligence.

Contributory negligence

In some cases a patient may by his own negligence contribute to his injury and formerly a plaintiff's claim was defeated if the defendant proved contributory negligence. The law has now been amended by the Law Reform (Contributory

Negligence) Act, 1945, which makes liability in cases of contributory negligence apportionable between plaintiff and defendant. Where any person who suffers damage as a result, partly of his *own fault, and partly of the fault of another, a claim in respect of that damage shall not be defeated* by reason of the fault of the person suffering the damage, but the damages in respect thereof shall be reduced as the Court thinks just having regard to the claimant's share in the responsibility for the damage.

Even so it is wise to keep notes describing the exact sequence of events, since although doctors are no longer likely to *defeat* a plaintiff's case against them (by establishing contributory negligence) they may secure *mitigation* of damages.

Should the patient refuse to obey instructions in an emergency following a medical accident, the practitioner would be wise to cease treatment, and thus, by establishing contributory negligence *weaken the patient's claim against him*. But he would be well advised to give the patient full warning of his intention and of his reasons for it and also to give him reasonable opportunity to follow his instructions. He should, in fact endeavour to provide what the Court would regard as clear and ample evidence that he had done everything that an ordinary and reasonably skilful practitioner would be expected to do.

PERMISSION TO ACT IN AN EMERGENCY

The patient, or in the case of a child, the parents, should always be consulted and permission obtained before carrying out any possibly hazardous procedure other than what is needed *in the most sudden and dire emergency*. Written permission is not essential but in any subsequent proceedings it provides better evidence that consent was given. If the parents cannot be consulted in time, the authority of the headmaster or whoever stands *in loco parentis* should be sought. A full statement of what has been done should be communicated to the parents as soon as possible.

DYING DEPOSITIONS AND DECLARATIONS

Although there is no legal obligation on a doctor to take down a statement by a dying patient, he is usually the best person to do this in an emergency. Two situations must be clearly *distinguished* —

(1) Where the *doctor* thinks the patient is unlikely to recover but the patient is unaware of the imminence of death. In this case a **deposition** should be taken. A magistrate should be summoned and it will be his responsibility to see that legal requirements are complied with *e.g.* that an accused person or his legal representative should have the right to cross examine. Such a deposition properly taken is admissible in evidence.

(2) Where the *patient* is in settled hopeless expectation of death. Here a **dying declaration** is made by the patient. It should be written down together with any questions used to elicit full information and their answers. A phrase such as 'Having the fear of death before me and being without hope of recovery' must be included. The presence of a magistrate is unnecessary. The declaration should be signed by the dying patient (if possible) and the person who writes it down. Witnesses are not necessary but are desirable. This type of declaration is limited to charges of homicide and is not admissible as evidence if referring to homicide other than that of the declarant.

CRIMINAL ABORTION

The obligation on a doctor or any third party to report a crime depends on whether the crime is a felony or a misdemeanour. Misprision (concealment) only becomes a crime in the case of felonies. The offence of concealment of a felony is almost obsolete and a prosecution is most unlikely.

If a doctor contemplates giving information to the police about an abortion he should seek expert legal advice since there is no certainty that he will be protected against subsequent litigation for defamation.

If the patient is dying the doctor may urge her to make a statement but he should not put leading questions. When she *dies* the *Coroner must be informed*. If *criminal abortion* is only suspected and the patient recovers there is no obligation to make any report.

Especially during wartime it sometimes happens that the *returning husband finds his wife ill*. When this illness is caused by abortion and it is clear that the husband could not have been responsible for the pregnancy the doctor should be careful of his replies to the husband's questions. The making of a defamatory statement to a husband about his wife is publication and

could render the person making it liable to an action for slander. Such an action could not of course succeed if the statement giving rise to it were true but if the doctor is not sure of his facts he should not say anything and would be wise to let the wife tell the husband herself.

DEATH BED WILLS

We have to distinguish between witnessing and helping to make a death bed will.

Anyone who understands that he is witnessing a signature may witness a will. It is not necessary that the witness should know that the document is a will. He is attesting the signature and is not concerned with the patient's mental state or how the patient disposes of his property. The doctor who attested a will may of course be called as an expert witness and asked if he thought the patient was sane when he signed his will.

The practitioner should only help the patient to *make* his will in an emergency and when a solicitor cannot be found. He must be satisfied that the patient is of sound disposing mind.

If previous wills have been made it is advisable to begin by revoking these. In most instances marriage automatically revokes a will. All names and addresses of possible devisees and legatees and descriptions of bequests should be full and accurate and any possibly ambiguous words such as *money* should be avoided. If illegitimate children are to benefit they must be named.

Reference to sums of money should be followed by the words *free of duty* and the residue of the testator's property should be described as *all the remainder of my estate real and personal*.

The testator should sign the will if possible or make a mark. If he is quite unable to do either then someone may sign in his place but the testator should if possible acknowledge in some way as by nodding his head that he understands what is being done. If a third person does sign on the testator's behalf he *must* sign in the presence of the testator and the testator must be shown to have seen the signature. Two or more witnesses must sign the will *in the presence of the testator* (this is essential) and preferably *in the presence of each other and nothing*

more must be added. The following sentence is usually put at the end of the will —

Signed and acknowledged by the said A B the testator as and for his last Will in the presence of us present at the same time who at his request in his presence and in the presence of each other have thereunto subscribed our names as witnesses

POWER OF ATTORNEY

If a doctor sends his patient to a mental hospital he may be asked what happens to the patient's money. He should advise the relatives to consult a solicitor but it is helpful to be able to say what the procedure will be.

If the patient is not certified a power of attorney is adequate provided that the patient is capable of understanding it. This is simply a formal appointment of an agent by a deed. This is usually drawn up by a solicitor and signed by the patient. It usually runs —

Know all men by these presents that I A B of hereby appoint C D my true and lawful attorney in my name or otherwise and on my behalf to do and execute the following acts and deeds — (Then follows a description of what the agent is authorised to do.) In witness etc etc

If the patient is certified and the estate is of any size an application should be made by the patient's solicitor to the Master in Lunacy. The Law Courts W C 2 for the appointment of a receiver. In Scotland relatives or failing relatives any parties interested or the incapax himself may petition the Court for the appointment of a Curator Bonis.

THE FAILED SUICIDE

Attempted suicide amounts to a misdemeanour only and there is therefore no legal obligation upon the doctor to report the matter to the police. He should do this only in those cases which are likely to end fatally or for which adequate arrangements such as care by relatives cannot be made. In all cases the doctor should do what he thinks best. If he does not think that a prosecution would be in the patient's interest he should not report the case.

ALCOHOLIC INTOXICATION

Examination of a person suspected of being intoxicated should

be regarded as an emergency procedure because in such a case important evidence is evanescent. Having been taken into custody by the police the prisoner is examined by the Police Surgeon and is informed of his right to have his own doctor's opinion. He should not be examined without his free consent since without this an examination is technically an assault. If he is unconscious or inaccessible there is no objection to examining him on the ground that he cannot consent. Consent to any reasonable examination may be presumed.

All details of the examination should be recorded on the spot and should include the exact time of the examination and a note of the prisoner's insight into the nature of the situation. The doctor may consult with the Police Surgeon as he thinks fit. Three questions about the patient should arise in the doctor's mind —

- (1) Is he ill? i.e. could his present state be brought on by illness such as G.P.I. or by drugs such as insulin?
- (2) Is he drunk? i.e. is alcohol responsible for his state?
- 3) How drunk is he? i.e. is his ability to perform some special act such as driving a car impaired?

Since individuals react differently to alcohol according to their make up and state of health it is difficult to draw a sharp line between the sober and the drunk.

Ordinary physical examination should be made. In a person under the influence of alcohol the temperature is subnormal the pulse rapid and bounding the pupils dilated and sluggish and the tongue dry. The patient's breath smells of alcohol and his speech is slurred. He tends to gesticulate wildly and breaks down emotionally during conversation. Hiccup salivation drowsiness and confusion are also significant signs.

Certain special tests should be applied and in order to avoid the occasional case of improvement for one test due to alcohol several should be employed. These are —

- Walking along a chalked line
- Smoking a cigarette
- Reading and writing
- Using a telephone directory

Blood alcohol

A percentage concentration of absolute alcohol in the blood by volume of 0.15 is generally taken as a level of intoxication and its estimation may be useful though it is not current practice in Britain. Five c.c.m. of blood should be taken and oxalated though it is unwise to do this if the patient is unconscious. A note should be made of the amount and type of beverage and time when it was taken. Since the alcohol content of the urine corresponds to the average blood concentration during the time of its secretion it is wise to save a specimen of urine.

EMERGENCY ASPECTS OF DEATH

The Registrar of Deaths is the person whose legal duty it is to report to the Coroner deaths which seem to him to come within the Coroner's jurisdiction. In Scotland the officer corresponding to the Coroner is the Procurator Fiscal.

No such legal duty is placed on the doctor who is in the same position as any layman. Nevertheless it is mutually convenient for the doctor to report certain deaths to the Coroner (or Procurator Fiscal). These are

- (1) All cases of unexpected death of unknown cause
- (2) All cases of violent death. An operation is technically an injury and so a death due to an operation comes under this heading. A death which may have been due to an anæsthetic must be reported whether it occurred on the operating table or not.
- (3) All deaths of lunatics
- (4) All deaths of foster children
- (5) All cases where the doctor did not see the deceased within 14 days before the death.

If a patient should die on arrival at hospital before being seen by a qualified and registered medical practitioner it is best to ask the doctor who last saw the patient to issue a death certificate. If he cannot or will not the Coroner (or Procurator Fiscal) should be informed.

Post mortem emergencies

Even in the mortuary emergency situations may arise. An attendant once raised the alarm because he found blood dripping from the head of a corpse. Investigation showed that bleeding

originated in the severed jugular veins following a partial autopsy (brain only).

EMERGENCY BAPTISM

Any child which shows signs of life is qualified to be baptised even if it is not viable. It is not usual to baptise prematurely delivered products of conception which do not show signs of life. In deciding whether to baptise or not the wishes of the parents should be carried out.

Baptism may be administered by any adult male or female whether baptised or not and irrespective of religious belief. Two things only are necessary, (1) Invocation of the Holy Trinity and (2) the use of water. Lay baptism should be performed only in the case of necessity, but the reality of the necessity is for the person performing it to decide. Even if the urgency is not great the validity of the baptism is not affected provided the proper matter (*i.e.* water) and form of words are used. Godparents are not necessary for private or emergency baptisms.

The doctor's fingers moistened with water should touch the child's forehead while he says—"I baptise thee A in the Name of the Father, and of the Son, and of the Holy Ghost." This simple form is valid for Roman Catholics as for others, and is not afterwards to be repeated.

If there is any doubt particularly in the mother's mind as to whether baptism has been already performed or whether the child is alive the baptism can be made conditional by saying "If you have not already been baptised, and if you are alive, I baptise thee." The clergyman of the parents' denomination should be informed.

In the case of a moribund nameless foundling, it is for the doctor to decide whether to baptise or not. If he does baptise a surname should be chosen afterwards. A clergyman of the Church of England should be informed unless the circumstances strongly suggest alternative procedures, such as the discovery of the child in a Roman Catholic church. It is well to choose names which would not handicap or embarrass him should he survive. The Local Authority who are his guardians have the right to choose names but will usually accept those given at an emergency baptism. The birth is registered by the Director of Public Assistance of the local authority after application to the Registrar General.

C ALLAN BIRCH

CHAPTER XXIII

Practical Procedures

WEIGHTS AND MEASURES

IT has not been felt justifiable to use the Metric system alone but for the benefit of readers who prefer this system the following conversion table is included. It shows the approximate metric equivalent of the common doses in the Apothecary System.

It will be found more convenient however to prescribe the *nearest / metric dose* in the Metric system to the dose in the Apothecary system rather than the *exact equivalent*. For example while in the United Kingdom we prescribe morphine gr $\frac{1}{6}$ or $\frac{1}{4}$ (approximate metric equivalents 11 and 16 mgm) it is more usual in countries using the metric system to prescribe 10 or 20 mgm. Key equivalents in common use and convenient approximate equivalents are indicated in heavier print.

WEIGHTS

Apothecary or Troy		Metric	Apothecary or Troy		Metric
1 oz	30	gm	$\frac{2}{3}$ gr	40	mgm
4 dr	15	gm	$\frac{1}{2}$ gr	32	mgm
$2\frac{1}{2}$ dr	10	gm	$\frac{3}{8}$ gr	24	mgm
2 dr	8	gm	$\frac{1}{3}$ gr	22	mgm
75 gr	5	gm	$\frac{1}{4}$ gr	16	mgm
1 dr	4	gm	$\frac{5}{16}$ gr	13	mgm
40 gr	3	gm	$\frac{1}{8}$ gr	11	mgm
30 gr	2	gm	$\frac{1}{16}$ gr	8	mgm
15 gr	1	gm	$\frac{1}{32}$ gr	6.5	mgm
10 gr	0.65	gm	$\frac{1}{64}$ gr	5.4	mgm
$7\frac{1}{2}$ gr	0.5	gm	$\frac{1}{128}$ gr	4	mgm
7 gr	0.45	gm	$\frac{1}{256}$ gr	3.2	mgm
6 gr	0.4	gm	$\frac{1}{512}$ gr	2	mgm
5 gr	0.32	gm	$\frac{1}{1024}$ gr	1	mgm
4 gr	0.25	gm	$\frac{1}{2048}$ gr	0.65	mgm
3 gr	0.2	gm	$\frac{1}{4096}$ gr	0.54	mgm
$2\frac{1}{2}$ gr	0.16	gm	$\frac{1}{8192}$ gr	0.4	mgm
2 gr	0.13	gm	$\frac{1}{16384}$ gr	0.32	mgm
$1\frac{1}{2}$ gr	0.1	gm	$\frac{1}{32768}$ gr	0.26	mgm
1 gr	65	mgm	$\frac{1}{65536}$ gr	0.2	mgm
$\frac{3}{4}$ gr	50	mgm	$\frac{1}{131072}$ gr	0.1	mgm

LIQUID MEASURES

Apothecary	Metric	
1 pint (20 fl. oz.)	568	c.cm. (Approx. 600 c.cm.)
12 fluid ounces	340	c cm
8 fluid ounces	227	c cm
6 fluid ounces	170	c cm
4 fluid ounces	114	c cm
3 fluid ounces	85	c cm
2 fluid ounces	57	c cm
1 fluid ounce	28 4	c.cm. (Approx. 30 c.cm.)
4 fluid drachms ($\frac{1}{2}$ fl oz)	14	c cm
2 fluid drachms (120 minims)	7	c cm
1 fluid drachm (60 minims)	3 5	c.cm. (Approx. 4 c.cm.)
50 m	3	c cm
45 m	2 7	c cm
30 m	1 8	c cm
20 m	1 2	c cm
15 m.	0 9	c.cm. (Approx. 1 c.cm.)
10 m	0 6	c cm
8 m	0 5	c cm
5 m	0 3	c cm
3 m	0 18	c cm
2 m	0 12	c cm
1 m	0 06	c cm

THE USE OF THE TELEPHONE IN EMERGENCIES

In order to call the police or an ambulance the telephone should be used in accordance with the instructions issued by the Post Office

With the automatic (dial) telephones of London and certain other places, 999 should be dialled. This does not call the police or ambulance direct but operates a special signal at the telephone exchange. The call is then passed to the appropriate emergency authority by the operator according to the request of the caller. In some areas emergency calls are made by dialling "O" or "O1"

EXTRACT FROM RULES FOR THE BROADCASTING
OF S.O.S. AND SIMILAR MESSAGES

1. For relatives of sick persons.

The British Broadcasting Corporation (Broadcasting House,

London W 1 Telephone WELbeck 4468) will broadcast messages requesting relatives to go to a sick person only when the Hospital Authority or the medical attendant certifies that the patient is *dangerously ill* and if all other means of communication have failed. In the normal course of events messages will be broadcast only when the full name of the person wanted is available.

NOTE.—When the person sought is known to be on board a ship at sea a message can only be broadcast if the ship is not equipped with apparatus for the reception of messages by wireless telegraphy. Further there must be a possibility that the return of the person sought can be hastened by the reception of such a message. This is not considered to be the case when the ship is on its way to a known port. In such cases enquirers are advised to communicate with the owners or agents of the ship or with the port authorities.

In no case can an S O S be broadcast requesting the attendance of relatives *after death has occurred*.

2 Appeals for special apparatus, foods or medicines for treatment of rare diseases will be broadcast only at the request of major hospitals and after every other means of obtaining them has failed.

No message can be broadcast regarding lost animals or property except where there is danger to life as from the theft of dangerous drugs or from escaped wild animals and then only at the request of the police.*

(3) There is no charge for broadcasting S O S messages.

Position

LUMBAR PUNCTURE

The patient should lie on his left side with his buttocks and shoulders on the hard edge of the bed (Fig. 42). If the mattress sags put fracture boards under it. The long axis of the spine should be horizontal and the plane of the iliac crests vertical. The spine must be fully flexed and the patient should be asked to get the chin as near to the knees as possible. A roller towel placed round the neck and knees and tightened by twisting with a rod sometimes helps to obtain and maintain the flexed position.

*If a doctor loses his bag he should emphasise the fact that it contains dangerous drugs. This will expedite measures to recover it.

Site of Puncture

The usual site is the space between the spines of the 3rd and 4th lumbar vertebrae. This space is on a plane passing through the highest points of both iliac crests.



FIG. 42

Position for lumbar puncture. An assistant places one arm under the knees and the other round the back of the neck in order to flex the spine as fully as possible.

(See Surgical Handcraft)

The Puncture

If difficulty is expected a stout (Barker) needle or a nickel needle (see page 12) should be used. For routine use a smaller needle is better. Punctures must be made under full aseptic precautions. Everything should be dry including the operator's hands. Gloves are not essential and instead of them a sterile towel may be used through which the needle is held and the skin palpated.

A weal of 2 per cent procaine is raised in the skin over the junction of the lower and middle thirds of the interspace. The skin is pierced here by giving the lumbar puncture needle a rolling motion. The direction of the needle is then readjusted so that it is in the horizontal plane and inclined about 5 degrees in a cephalad direction corresponding to the slope of the vertebral spines. No resistance is felt until the ligamentum flavum is

reached. It is advisable to pierce this with the bevel in the spinal axis so that the fibres are separated rather than cut across thereby ensuring that the puncture will close more easily. The dura is

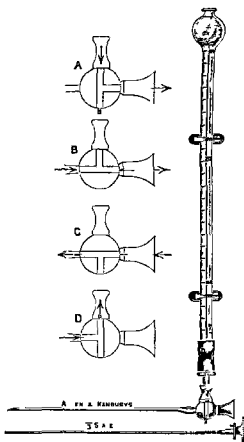


FIG. 43

Greenfield's spinal manometer

next encountered and penetrated. Withdrawal of the stylette allows C S F to flow.

Difficulties

- (1) No fluid flows. Advance the needle a few millimetres and rotate it in case a nerve root is obstructing it.
- (2) Bone is encountered. This usually means that flexion is incomplete. Withdraw the needle and adjust the position.

- (3) Blood appears If it is only a few drops and then nothing more it means that the needle has not gone far enough but is in the subdural space By over enthusiastic insertion one may encounter the anterior subdural space

If the fluid is bloodstained the decision must be made as to whether the bloody tap is due to trauma or subarachnoid

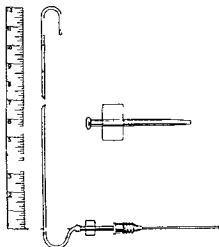


Fig 44

Northfield's apparatus for measuring the cerebro spinal fluid pressure

hæmorrhage Traumatic bleeding often clears as the fluid drains If in doubt take specimens in three numbered tubes and have red cell counts done on all three A diminishing count indicates traumatic blood

Clotting in the fluid may occur if bleeding was due to trauma and if centrifugalised the supernatant fluid is colourless In subarachnoid hæmorrhage clotting does not occur and the supernatant fluid is yellow

Measuring the C S F pressure

This cannot be done by observing the rate of flow A manometer such as Greenfield's (Fig 43) must be attached to the needle A simpler method is to insert into the needle a butterfly adaptor with a length of bicycle valve tubing attached and at the other end of which is a small glass U tube This is held below the level of the needle until fluid appears in it and then

raised until the fluid in one of its limbs is steady. A metal ruler with its zero mark level with the needle serves to read the pressure (Fig. 44)

Routine study of dynamics

Record the resting pressure and its response to coughing and straining. Failure to respond means that the needle is not in the correct place. Compress the right and left and both internal jugular veins and record results (Queckenstedt's test)

The Dattner (double) needle

This is useful if lumbar puncture has to be performed in cases of raised intracranial tension and also to minimise the risk of post puncture headache.

The device (Fig. 45) consists of a fine needle and stylet inside a larger needle. The outer needle is inserted down to the ligamentum flavum and the inner needle punctures the dura. If used carelessly the outer needle may penetrate the dura and the advantage of the Dattner's principle will be lost. If correctly performed fluid should cease to flow when the inner needle is withdrawn into the outer case. A syringe must be used to withdraw C.S.F. Manometry is not possible.

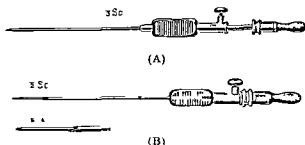


Fig. 45

Dattner's needle

(A) Arranged for introduction

(B) with needle projecting after introduction

CISTERNAL PUNCTURE

Position

The patient should lie with his head at the foot of the bed so that the bedpost is not in the way. The head is supported

on a small sandbag and flexed. The spine should be horizontal (Fig 46)

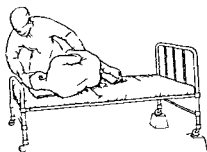


FIG 46

Position for cisternal puncture

Site of puncture.

This is determined by the fact that a horizontal plane through the tips of the mastoid processes bisects the atlanto occipital space (Fig 47). The tips of the mastoid processes are marked and a

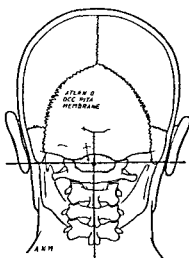


FIG 47

The point of entry of the needle at the intersection of a line joining the tips of the mastoids and the vertical mid line of the neck

(*P. J. Surg. and H. H. Croft*)

horizontal line joining them is drawn using a tape measure and a grease pencil. The point where this line crosses the vertical mid line of the neck is marked. This is the entry point.

The puncture

A Purves Stewart graduated needle (Fig 48) is convenient but a lumbar puncture needle may be used. A scratch made on it 5 cm. from the tip is a useful guide.

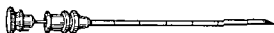


FIG 48

Purves Stewart's cisternal puncture needle
(*The Surgical Handcraft*)

The needle is introduced horizontally kept strictly in the mid line and advanced cautiously (Fig 49). A characteristic feel is experienced as the atlanto occipital ligament is penetrated. After a further advance of one or two millimetres the stylette is withdrawn and fluid appears. Some operators prefer to mount the needle on a syringe and apply slight suction when the atlanto occipital ligament is pierced.

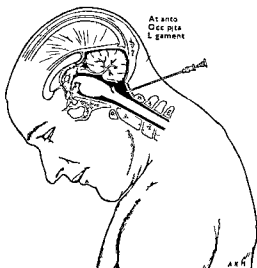


FIG 49

Showing the direction of the needle which has reached the atlanto-occipital ligament
(*Singer of Modern Warfare*)

Because of the risk of damaging the medulla by penetrating too far it is useful to determine the depth at which the cistern may be reached. This is done by measuring how far in a sagittal

plane the entry point is behind the vertical plane through both mastoids. This "skin mastoid" distance is the shortest distance from the tip of one mastoid to a transverse line through the entry point. To measure it one limb of a folding ruler is placed transversely over the entry point and the other limb at right angles to it is placed antero posteriorly under the lobule of the ear. The

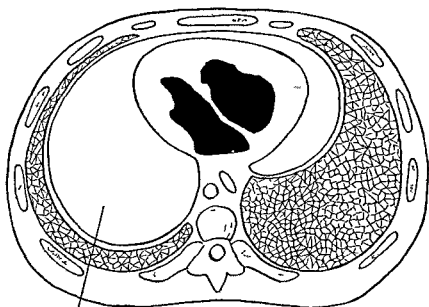


FIG. 30

Diagram of Percardial tapping by the posterior route

distance of the mastoid tip to the transverse limb can then be read off. The average distance is 7 cm. Subtraction of 2.5 cm gives the depth of the atlanto occipital ligament. The ligament and dura are about 0.5 cm thick and so subtraction of 2 cm gives the depth of the cistern. It is roughly 2 inches from the skin and the medulla is about half an inch further on.

PARACENTESIS OF THE PERICARDIUM

There are three possible routes—anterior, epigastric and posterior.

- (1) **Anterior** The needle is inserted (a) in the fifth left interspace just outside the apex beat but inside the outer edge of the cardiac dulness or (b) in the fourth left interspace one inch from the sternal margin (to avoid the internal mammary artery)

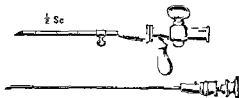


FIG 51
Rivière's needle

- (2) **Epigastric** The needle is inserted in the angle between the ensiform cartilage and the costal margin and directed upwards at an angle of 20° to the chest surface to enter the lowest part of the pericardial sac
- (3) **Posterior** The needle is inserted near the inferior angle of the scapula on the left side. It traverses the lung to reach the pericardium

The skin is cleansed and the skin and subcutaneous tissues infiltrated with 2 per cent procaine. A long needle of about 1 mm bore is used for the pericardial tap. The anterior routes are the safest but it is not possible to remove large quantities of fluid by them. When an effusion is very large it collects posteriorly and compresses the lung (Fig 50). In such a case the posterior route is best. When empyema of the pericardium is suspected approach through the pleura and lung is contra-indicated and the epigastric route using a fine trocar and cannula is to be preferred. It is probably wisest in such a case to enlist the help of a surgeon.

TECHNIQUE OF ARTIFICIAL PNEUMOTHORAX INDUCTION

In young or sensitive patients sedation is advisable and *inctus* of codeine phosphate should be given if there is a cough.

The patient lies on the good side with his upper arm raised to expose the axilla. With aseptic precautions the skin and tissues down to the pleura are infiltrated with 2 per cent

procaine in the fourth fifth or sixth inter costal spaces in the mid axillary line

A Rivière's needle (Fig 51) attached to an artificial pneumothorax apparatus and open to the manometer is inserted down to the pleura. It is best to enter it at the upper border of a rib so as to avoid the vessels in the sub costal groove of the rib above. The sharp trocar is then removed and the cannula pushed through the pleura. A good negative swing of the manometer fluid in both phases of respiration indicates that the needle is in the pleural space. The clips are then turned on and air is drawn into the chest. The usual amount for induction is 100 to 300 c cm.

Failure to enter the pleural space is shown by the behaviour of the manometer —

- (1) A small equal swing on each side of zero means that the needle is in the lung tissue. A similar but larger swing indicates that it is in a lung cavity.
- (2) A rising positive pressure means that the needle has punctured a blood vessel. It should be withdrawn promptly.
- (3) Absence of any reading means that the point of the needle is in the chest wall, fluid, or an adhesion, or that the tubing or needle is blocked. But a small negative swing of the order of 1-3 is sometimes obtained when the needle point is situated extra pleurally.

In the case of failure other sites should be tried before abandoning the procedure, such as the 7th or 8th spaces posteriorly and the 2nd, 3rd and 4th spaces anteriorly.

Numerous types of apparatus exist, one of the most popular being the Lillingston Pearson apparatus consisting simply of two bottles and a manometer (Fig 52). When using an apparatus of this type for an induction the fluid levels in each bottle should be at the same height. The negative intra pleural pressure draws air into the chest. If the fluid levels are different, air enters the chest under pressure and the risk of air embolism is increased.

TECHNIQUE OF PNEUMO PERITONEUM INDUCTION

The most convenient site for the intra peritoneal injection of air is near the left subcostal margin just external to the rectus abdominis muscle.

With aseptic precautions a wheal of 2 per cent procaine is raised in the skin with a small hypodermic needle. The syringe (2 c cm)

is recharged and using a long serum needle the abdominal wall is infiltrated down to the peritoneum. When this is punctured the sense of resistance to the piston disappears. Sudden penetra-

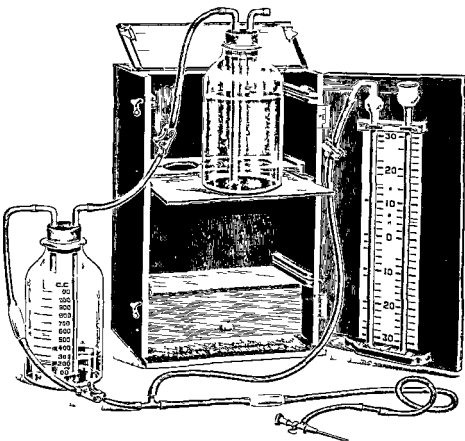


FIG. 57

Lillingston Pearson artificial pneumothorax apparatus

tion should be avoided. The piston should be withdrawn to make sure that the needle point is not in a blood vessel. A pneumothorax apparatus is then attached to the needle by an adaptor. Air is slowly injected in amounts up to 1 000 c.c.m.

A manometer is unnecessary but if available provides a useful indication that the needle is in the correct space by showing

respiratory excursions and also a rise when the abdomen is pressed on. If before the air is run in the clips are suddenly opened and closed the fluid in the manometer will rise and fall rapidly to almost zero if the peritoneal space has been entered. If the needle is outside the peritoneum the pressure will remain high.



FIG 53

Posterior splanchnic block Position of entry point
(Lambert)

Some operators will prefer a pneumothorax needle and others a special Veress needle. This consists of a sharp needle in which runs a hollow blunt ended trocar with a lateral hole. The trocar is made to project slightly from the needle by a spring thus ensuring that the gut is pushed away by the blunt end rather than the sharp needle point.

Clinical evidence that air has entered the peritoneal space is provided by —

- (1) Pain in the shoulder regions (but not on the side on which the phrenic nerve has been interrupted)
- (2) Disappearance of liver dullness

The most frequent cause of failure to induce a pneumoperitoneum is that the needle is not inserted far enough.

POSTERIOR SPLANCHNIC BLOCK

The patient lies on his side supported by pillows so that his spine is horizontal. After anaesthetising the skin a 12 cm needle with a stylet is inserted 7 cm from the mid line at the level of the spine of the first lumbar vertebra. This point is just below the 12th rib (Fig 53). It is directed anteriorly and medially at an angle of 45° to the sagittal plane until it strikes the body of the 1st lumbar vertebra. It is then withdrawn a little and

manipulated until its point is felt to slide tangentially past the body of the vertebra after which it is advanced 1 cm (Fig 54)

A negative aspiration test being obtained the anaesthetising solution is then injected. This may be 25 c cm of one per cent

VERT

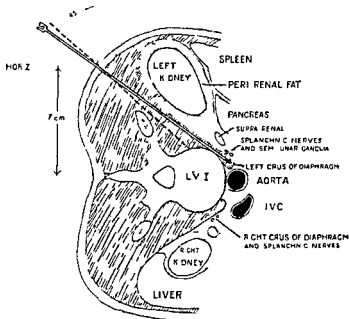


Fig 54

Posterior splanchnic block Direction of needle
(J. J. Hauer)

procaine with adrenaline 0.0005 per cent or 50 c cm of 0.05 per cent (1 in 2000) butethanol (amethocaine) with adrenaline 0.0005 per cent. This procedure may be repeated on the opposite side.

TIDAL IRRIGATION OF THE PARALYSED BLADDER

The principle is that the bladder is slowly filled with sterile normal saline or a mild antiseptic solution from a container. When the intra vesical pressure reaches a predetermined level the bladder is emptied by siphonage and the cycle starts again.

Intra vesical pressure is measured by the height of fluid in a

cystometer tube. By measuring the pressure after each successive 50 c cm have been run in, a cystometrogram is constructed. In this way the tone of the bladder and its reflex contractions can be observed. As the tone improves the intra vesical pressure rises more steeply in response to the volume of the bladder contents.

- A Reservoir
- B metal screw shutting off clamp
- C Murphy drip bulb (air vent in side closed)
- D glass tube with end drawn out to pin point opening connected to the top of the air vent tube by a short rubber tube
- E $\frac{1}{4}$ inch glass tubing to serve as manometer for measuring intravesical pressure
- F scale
- G air vent tube (capillary tubing with inside diameter approx $\frac{1}{16}$ inch)
- H metal clamp to shut off manometric tube when apparatus is in use as tidal irrigator
- J standard Dakin's glass tube with four outlets
- K siphon tubing
- L collecting bottle
- M metal screw-clamp to cut out siphon when doing cystometrograms
- N loop of cord supporting siphon tubing

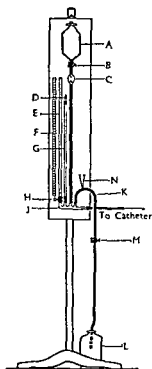


FIG 55
Tidal irrigator and cystometer combined

When reflex contractions are sufficiently strong tidal drainage can be discontinued and the bladder allowed to empty itself reflexly. If the residual urine after the bladder has emptied itself is found to be large, tidal irrigation will need to be started again until reflex evacuation is more complete.

"Operational details" (Fig. 55.)

The fluid from the drip enters the horizontal arm of the glass Dakin's tube (J) and passes into the bladder through the rubber tubing connected to the indwelling catheter. It also ascends into the air vent (G) and into the siphon tubing (K) as the intravesical pressure rises. When the pressure within the bladder rises

sufficiently to force the column of fluid over the syphon tube loop drainage of the bladder begins by siphonage. The column of fluid in the air vent roughly parallels that in the syphon tube loop. When siphonage begins fluid is drawn out of the air vent tube at once and air is sucked in through the pin point opening (D) but too slowly to break the siphon until the bladder is empty. When the bladder is completely empty the cycle begins again. It is always possible to tell at a glance whether the apparatus is working properly for respiratory excursions can be seen in the column of fluid in the air vent tube. With any block between the system and the bladder these disappear. The indwelling catheter should be changed every three or four days. The patient should have the workings of this apparatus explained to him and be charged with noting the respiratory excursions and the rate of drip of the irrigating fluid.

For tidal irrigation the sterilised apparatus is assembled on a board to which it is attached by ties passing through holes in the board. The flask containing the irrigating fluid is suspended at least 75 cm above the level of the Dakin's tube. The board is then suspended so that the Dakin's tube is level with the patient's symphysis pubis. The desired height of the syphon tube loop is secured by suspending it with cord passed through holes in the board. Before the apparatus is connected to the indwelling catheter fluid should be run through it to eliminate air. The apparatus is connected to the catheter by a glass connecting tube which should have a lumen at least $\frac{1}{4}$ inch in diameter. The clamp (B) is regulated to allow the fluid to drip at the desired rate. The number of drops per minute depends on the number of times the bladder is to be irrigated and by what quantity of fluid. For example if it is desired that the bladder is to be evacuated when 300 c cm have been run in and this is to be done 10 times a day the rate of delivery is 125 c cm per hour or a little over 2 c cm per minute and the drops per minute would be about 50. In practice this calculation must take into account the secretion of urine into the bladder which goes on continuously. If infection is present when tidal irrigation is begun the number of daily irrigations should be increased and therefore the rate of drip speeded up. This is true also if at any time during convalescence symptoms of infection develop. Frequent irrigations will quickly clear the urine

and the symptoms of toxicity. If symptoms of infection do occur during the period of tidal irrigation they usually mean that the apparatus is not working properly and the cause should be looked for and corrected.

The height of the siphon loop is that which the intra vesical pressure must reach to initiate siphonage. If the bladder is to be emptied when 300 c cm of fluid have been run in the pressure within the bladder produced by this amount of fluid must be known so that the siphon loop can be set at this level. A cystometrogram is always done before tidal irrigation is begun to determine the tone of the bladder musculature and the level at which the siphon loop is to be set. Regular cystometrograms taken at short intervals are essential for regulating the rate of drip and the siphon tube height for as the bladder tone improves readjustment in both must be made.

For cystometry it is only necessary to close off the siphon tube by means of the clamp (M) and loosen the clamp (H) to admit fluid into the manometer tube for intravesical pressure readings. With a glass Dakin's tube at the level of the symphysis pubis the height of the column of fluid in the manometer measures the intravesical pressure. The bladder should be empty before starting the cystometrogram and fluid should drip into the bladder at about 120 drops per minute. Readings are usually taken at 50 c cm intervals. Bladder contractions are plotted whenever they occur. The cystometer incorporated into this combined apparatus is the same in principle as the one used by Munro. It may be possible to simplify this combination even further. The one described here was useful in the management of war casualties.

For more detailed study of the bladder reactions and behaviour a glass T tube may be interposed at the junction of the manometer tube (E) and the arm of the Dakin's tube. The T tube is connected to a tambour and writing pen which records continuously on a moving drum all changes of intravesical pressure.

Supra pubic route.

To avoid the disadvantages of the indwelling urethral catheter of the above method on the one hand and a supra pubic cystotomy on the other, tidal drainage may be applied through a

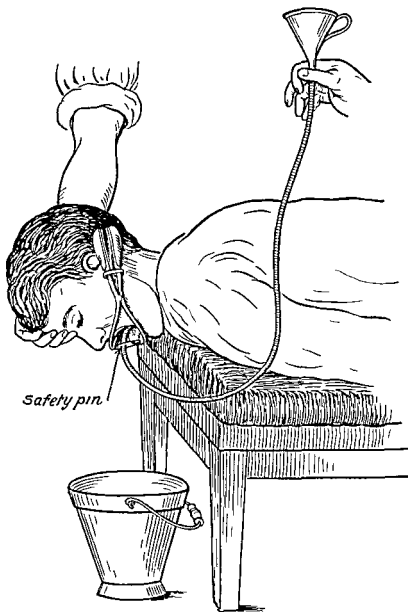


FIG 56

Gastric lavage showing the proper position of the patient. For the sake of clarity the operator's hand holding the tube just outside the patient's mouth and the assistant's hand holding the gag are not shown in the drawing. The head must on no account be raised higher than shown. (Dr H I Marriott)

small leakproof supra pubic catheter This requires a special technique for its introduction *

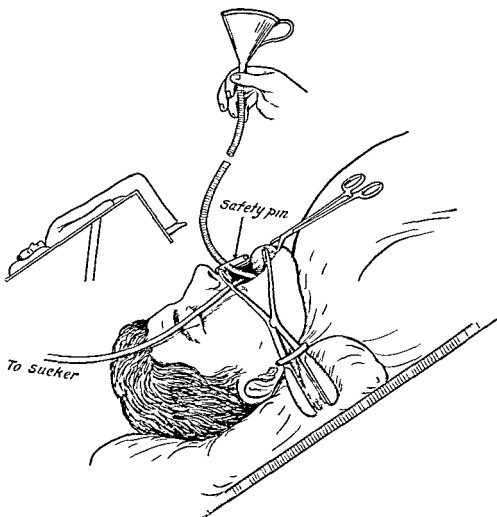


FIG 57

Gastric lavage using an operating table Hands holding the tube gag and tongue-clip are not shown (Dr H I Marriott)

GASTRIC LAVAGE

Position of patient

The mouth must be on a lower level than the larynx so that fluid cannot run into the trachea and bronchi This is very

* RICHES E W *Lancet* 1943 1 193

important particularly in the comatose patient who can easily be drowned by attempted gastric lavage if the head is raised

The patient should therefore be placed prone with his head

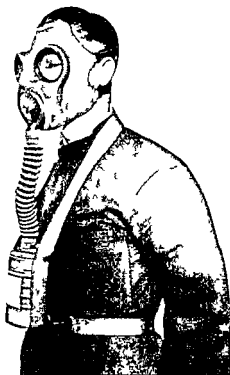


FIG 58
Puretha respirator
(See also Fig 111)

over the edge of the table (Fig 56) or in the Trendelenburg position on an operating table (Fig 57). In the latter position it is necessary to draw the tongue forwards by a clip and to remove fluid from the mouth by a sucker. A struggling patient may have to be immobilised by straps or by wrapping him tightly in a blanket.

Technique

Dentures are removed and the mouth opened by a gag. A fairly stiff oesophageal tube preferably 60 inches long and about

half an inch in diameter (for an adult) is lubricated and then passed over the tongue and quickly down the oesophagus. The end of the tube should be 20 inches from the incisor teeth in an adult and 10 inches in an infant. It is an advantage to mark these

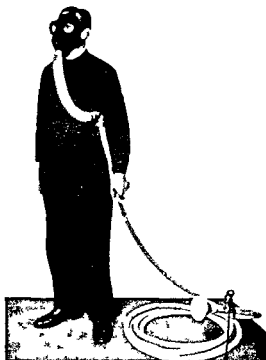


FIG. 59

Antipoy's short distance breathing apparatus
(Noble Gorman and Co. Ltd.)

distances on the tube by a safety pin in its wall (but not in its lumen)

As soon as the tube enters the stomach warm water is poured into a funnel attached to its end. After a pint has entered (less in children) but while the level of water is still visible in it the funnel is lowered over a pail on the floor and the fluid syphoned out of the stomach. A total amount of two gallons should be used and the first washings should be preserved in case analysis is required.

RESPIRATORS

Three main types are available for rescue purposes. At least

two models appropriate to the industry concerned should always be provided and workmen should be practised in their use

- (1) Canister types such as the Puretha (Fig 58) These can be relied on for short periods in low concentrations of gas. Numerous canisters of distinctive colour are provided to give



FIG 60
Salvus self contained oxygen
breathing apparatus
(S. de la Roche and Co. Ltd)

protection against different gases. Appropriate canisters should be available in any given industry.

- (2) Fresh air apparatus. The Antipoyss (Fig 59). This has the same facepiece as the Puretha respirator and is connected to an air pipe 30 feet long the end of which must be in fresh air.

- (3) Self contained oxygen apparatus *The Salvus* (Fig 60)
 This apparatus enables the wearer to remain up to half an hour in an atmosphere of 100 per cent irrespirable air. It delivers oxygen automatically at 2 litres per minute and there is a relief valve to deflate the breathing bag should this become overinflated. Eye protecting goggles should be worn also in smoke or gases affecting the eyes.

ARTIFICIAL RESPIRATION

Sudden failure of respiration demands prompt treatment for if breathing has stopped for ten minutes death is almost certain and may even occur after a two minute stoppage.

There are three main manual methods of performing artificial respiration —

- (1) For prone patients (Schafer)
- (2) For supine patients (Silvester Schuller)
- (3) *Rocking method* (Eve)

No one method is universally applicable and the choice will depend on which one the operator knows whether he is single handed or not and how much space is available. Success depends on the time factor and so the best method is whichever the operator can promptly and persistently apply. In all cases of drowning the patient should be inverted first.

Schafer's (face down) method (Figs 61 62)

Since few doctors know this method well the following extract is given from instructions prepared by Cecil K. Drinker M.D. Professor of Physiology Harvard School of Public Health

“ FOLLOW THESE INSTRUCTIONS EVEN IF THE PATIENT APPEARS DEAD ”

As soon as possible feel with your fingers in the patient's mouth and throat and remove any foreign body (tobacco false teeth). If the mouth is tight shut pay no attention to it until later. Do not stop to loosen the patient's clothing but immediately begin actual resuscitation. Every moment of delay is serious.

Proceed as follows —

- 1 Lay the patient on his belly one arm extended directly over head the other arm bent at elbow and with the face turned

outward and resting on hand or forearm so that the nose and mouth are free for breathing (Fig. 61)



FIG. 61

Schäfer's method. Expiration is produced by the operator's weight on the shoulders, pushing up the diaphragm.
Expiratory effort on expiration



FIG. 62

Schäfer's method. Inspiration occurs while pressure is taken off the diaphragm.
Effort of inspiration on expiration

7. Kneel straddling the patient's thighs with your knees placed at such a distance from the hip bones as will allow you to assume the position shown in Fig. 61.

Place the palms of the hands on the small of the back with

fingers resting on the ribs with the thumb and fingers in a natural position and the tips of the fingers just out of sight (Fig 61)

3 With arms held straight swing forward slowly so that the weight of your body is gradually brought to bear upon the patient. The shoulder should be directly over the heel of the hand at the end of the forward swing (Fig 61) Do not



FIG 63
The Panis resuscitator

bend your elbows. This operation should take about two seconds.

4 Now immediately swing backward so as to completely remove the pressure (Fig 62)

5 After two seconds swing forward again. Thus repeat deliberately 12 to 15 times a minute the double movement of compression and release a complete respiration in four or five seconds.

6 Continue artificial respiration without interruption until natural breathing is restored if necessary four hours or longer or until a physician declares the patient is dead.

Inspiration should take three seconds (say one thousand two thousand three thousand or one chimpanzee and so on) and expiration two seconds.

The important point in Schafer's method is that expiration depends on compression of the abdominal viscera pushing the diaphragm upwards. The operator should feel that the loins are yielding. His hands should therefore be placed low down over the small of the back and not over the rigid part of the thorax. Expiration is effected by the elastic recoil of the thorax and diaphragm. The disadvantage of Schafer's method is that the operator cannot see what is happening. If there is an assistant he should determine by feeling and listening that air is going in

and out. Also if the patient is deeply unconscious and cold the diaphragm will be toneless and unable by elastic recoil to cause inspiration. Relays of operators are necessary as the method is tiring.



FIG. 64

Silvester's method (Inspiration). The shoulder pad is big enough to let the head fall back so that the tongue need not be pulled out. In this subject Schäfer's method was not possible because the abdomen did not touch the floor (waist 23 inches).

(See Artificial Respiration Explanatory)

The Panis Resuscitator (Fig. 63) is designed to apply Schäfer's prone pressure method mechanically. The patient lies prone on an inclined platform with a broad strap round the base of his thorax. A lever when depressed compresses his chest and inspiration occurs when springs release the lever.

Silvester's (face upwards) method (Fig. 64)

The patient lies face upwards with a pad under the shoulders large enough to let the head fall back. Kneel at the patient's head and induce inspiration (three seconds) by grasping the forearms and drawing them upwards and outwards until they are parallel to the side of the head and the elbows nearly touch the ground. Expiration (two seconds) is achieved by flexing the elbows and bringing the arms forwards and downwards and pressing them against the chest. Extra persons are usually needed and can assist by pulling out the tongue, pressing on the abdomen and by taking one arm each.

Schuller's rib traction method (Fig 65)

This is less tiring than the previous methods and is said to be more effective. The operator kneels at the head and faces the feet. He hooks his fingers round the costal margins which are pulled upwards and outwards steadily for three seconds and pressed downwards and inwards for two seconds.

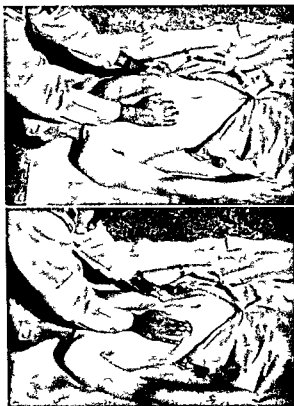


FIG 65

Schuller's rib traction method

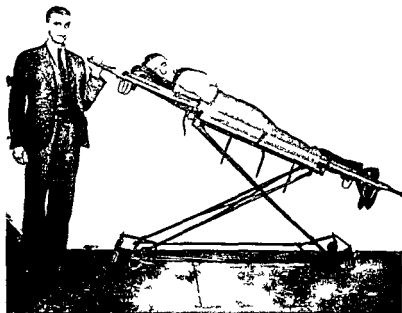
Eve's rocking method

Eve has pointed out that manual methods of artificial respiration depend on the elastic recoil of the thoracic wall and diaphragm for inspiration and that this may be absent in the drowned because of loss of muscular tone. In such cases a rocking method is advisable. Besides causing efficient ventilation it has the advantage of aiding circulation. By using a subject anaesthetised to the stage of respiratory arrest and

measuring the pulmonary exchange it has been shown that this was greater with Eve's rocking method than with the methods of Schafer or Silvester. Wet clothes can be replaced and dressings applied while rocking goes on. Relays of trained operators are not needed.

Rocking may be achieved in various ways —

- 1 On specially designed stretchers or beds (Fig 66)



F c 66

The To Tilter apparatus

- 2 On improvised rocking stretchers

A door or ladder seven feet long is obtained and the patient lashed to it. For a trestle a low fence or the backs of two chairs or a loop of rope may be used—all of which should be at least 34 inches high. A small two wheel handcart serves admirably.

Schafer's method is continued until the patient is securely strapped to the stretcher. Start with a full head down tilt and maintain it until no more fluid runs out of the mouth.

and then start rocking. The rate should be nine times a minute (four seconds head down and three seconds feet down). The angle should be 45° at first and may be reduced later to 30° .

3 Manually

This is illustrated in Figs. 67, 68, 69 and 70 from *Eve's Artificial Respiration Explained* but the grip for adults need-



FIG 67



FIG 68

Fig 67 Manual rocking Expiration. A single operator rocks a child (4 stone) and can walk to shelter while doing so.

Fig 68 Manual rocking Inspiration. As the abdomen is unsupported it sags the abdominal contents fall into the sag and this pulls down the diaphragm for inspiration.

(*See Artificial Respiration Explained*)

explaining. Two operators stand facing and each places an arm across the front of the patient's chest and grasps the patient's opposite axilla. Each passes his other arm under the patient's near thigh and grasps his opposite thigh. A third operator can assist by raising and lowering the legs.

General measures

As Eve points out, recovery depends on the circulation of warm oxygenated blood to the brain. Artificial respiration is not therefore merely a question of getting air in and out of the lungs.

but is a triad of warmth circulation and ventilation. Therefore without interrupting the rhythm of whichever method is used steps should be taken by assistants to keep the shocked and cold patient warm and to give him an analeptic injection (nikethamide 2 c cm)



FIG 69

FIG 70

Fig 69 Manual rocking an adult (10½ stone) Expiration

Fig 70 Manual rocking an adult (10½ stone) Inspiration This shows the Krill thigh grip with a forearm under each thigh and a hand grip over the outer thighs. The two operators are walking sideways to shelter during rocking each step counting a second.

(See first of Respiration Explanations)

Artificial respiration must be kept up until natural breathing is permanently restored or death occurs. In asphyxia from drowning and electrocution the usual signs of death are misleading and the patient should not be presumed dead until cooling rigor mortis or post mortem staining are evident.

The danger of all methods of artificial respiration is that by their over vigorous use CO_2 is washed out of the blood (acapnia) and the stimulus to normal respiration is removed. The rate by Schafer's and similar methods must not exceed 12 cycles per minute and by the rocking method nine per minute.

Artificial respiration on board ship.

Artificial respiration must be started in the rescue boat. Otherwise the patient may be dead by the time the shore is reached. Schäfer's method (not forgetting the initial complete inversion) may be tried if there is sufficient flat space.



FIG 71



FIG 72

Fig 71 Manual rocking in rescue boat Inspiration Two operators (facing astride a thwart) Their forearms support the mid thighs and upper chest of the victim

Fig 72 Manual rocking in rescue boat Expiration Arms and head dangle The hands grip the thighs

(See Artificial Respiration Explained)

It may be preferable and sometimes imperative to use the rocking method (Figs 71 and 72). If the patient is too heavy or the journey too long for manual rocking, then Silvester's method is best for use in a small boat (Fig 73). An improved rocking method on board ship is illustrated in Fig 74.

THE "NOVITA" RESUSCITATION APPARATUS

This apparatus is designed for administering "Dicarbox gas (93 per cent oxygen with 7 per cent carbon dioxide) in conjunction with one of the methods of artificial respiration

already described. The following notes are made by kind permission of the makers Messrs Siebe Gorman & Co. Ltd.

The apparatus (Fig. 73) comprises the following parts—

Two cylinders (A) (A) each of 15 cubic feet capacity at a pressure of 120 atmospheres.



FIG. 73

Siebe Gorman's method of resuscitation.
The victim lies face up on the knees
of the bearers, the head is supported
straight and the head hanging back.
Inspiration and expiration is
effected by No. 1
Efficient Positive Expiration

The cylinders are provided with shut off valves (B) (B) and are connected by a manifold (C). At each end of the manifold (C) is fitted a non return valve which permits one of the cylinders to be removed from the apparatus without loss of gas from the other.

To the manifold is connected a pressure gauge (D) which shows the pressure of gas in the cylinder.

To the other side of the manifold is connected a pressure reducing and control valve (E) fitted with a flow meter (F) calibrated in litres per minute. The flow of gas is controlled by rotating the tap (G). Rotation in a clockwise direction increases the flow and vice versa. From the reducing valve gas passes via the pipe (H) to the metal connecting piece (I) to one limb of which is connected the flexible breathing bag (K) and to the other the non return inlet valve (L) and the flexible breathing tube (M). A spring loaded non return air admission valve (N) is fitted for the purpose described hereafter.

To the end of the breathing tube is secured the rubber face mask (O) This is provided with a non return outlet valve (P) and a variable air admission port (Q)



FIG 74

The rocking method in a ship. The bight of rope suspended from two hammock hooks serves as a fulcrum almost as well as a trestle

(From *First Aid in the Royal Navy* by permission of Controller H M Stationery Office)

The use of the "Novita" apparatus.

START ARTIFICIAL RESPIRATION WITHOUT DELAY

- (1) See that the control tap (G) is turned to its full extent in the anti clockwise direction
- (2) Open the valve (B) of one of the cylinders (A) (A)

- (3) Open the air admission port (Q) on the face mask (O)
- (4) Apply the face mask to the patient pressing it into position so as to make a good joint
- (5) Rotate the control tap (G) in a clockwise direction until the flow meter (F) registers 10 litres per minute

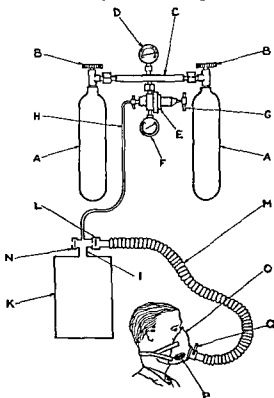


FIG. 75A

The Novita resuscitation apparatus
(From S. Cole (orman and Co. Ltd.)

- (6) Close the air admission port on the face mask
- (7) Watch the movements of the breathing bag (K). If this becomes over inflated reduce the flow of gas by rotating the control tap (G). If the bag becomes deflated increase the flow of gas
- (8) If the patient recovers to such an extent that he is consuming 30 litres per minute open the air admission port (Q) on the face mask and reduce the flow of gas to 25 litres. If recovery

is maintained reduce the flow and open the air admission port (Q) progressively increasing the flow if respiration appears to fail. The purpose of the spring loaded air admission valve (N) is to admit atmospheric air if by chance the patient should empty the breathing bag.

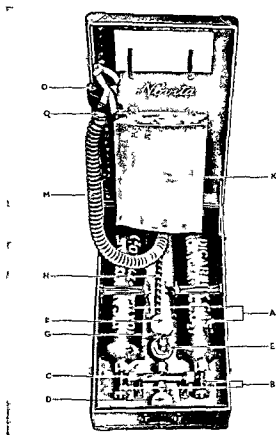


FIG 75B

The Novita resuscitation apparatus
(From Siebe Gorman and Co Ltd)

Breathing machines.

In cases where artificial respiration is likely to be necessary for long periods (*e.g.* poliomyelitis) a mechanically operated breathing machine is necessary. Because many hospitals now possess a Both Cabinet Respirator (Fig 76) thanks to Lord Nuffield the details of operating this machine are given. In the area of the Metropolitan Police the Emergency Bed Service

(Tele MONarch 2394) has a list of machines ready for immediate use. For description of other machines such as those which do not enclose the patient the reader is referred to *Breathing Machines* H M Stationery Office 1939

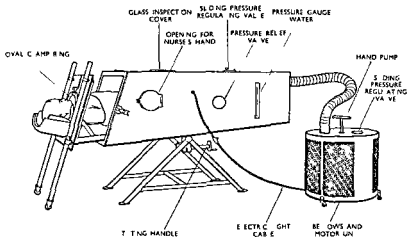


FIG 76
The Both cabinet respirator

The use of the "Both" respirator

(1) Test the respirator

Close the cabinet. Switch on the motor. Hold a pillow against the head opening. The manometer should show an oscillation of at least 10 cm of water. Absence of adequate oscillation means leakage of air.

(2) Prepare the respirator for the patient

Attach the rubber diaphragm to the head piece. Open the cabinet fully and put Sorbo mattress mackintosh draw sheet etc on the couch. Start the motor. If this fails prepare to use the hand pump as follows

HAND OPERATION IN AN EMERGENCY

- (1) Switch off the current and withdraw the plug. This is to ensure that the motor shall not be switched on again with the bellows disconnected.
- (2) Detach the rubber hose and remove the perforated metal cover.

- (3) Push the small loose bolt near the centre of the lower bellows disc, upwards into the bellows and hold it there
- (4) Pull out the pin with a knurled head which couples the connecting rod to the bottom board of the bellows
- (5) Slip the connecting rod out of its socket and replace the pin
- (6) Replace the handle and operate the bellows with the handle making 20 strokes per minute
- (3) Place the patient in the respirator

Three persons are required. Two standing on the same side lift him from the trolley to the couch of the respirator. The third person puts his hands through the rubber diaphragm and draws the patient's flexed head through aided by the pushing of the other two. Push back the couch and close the machine.

Support the head on pillows and place a collar of greased lint between his neck and the diaphragm. Extra cotton wool may be needed to prevent leakage. Unclamp the diaphragm and slip in behind and in front of it the thin metal templates and clamp up again. These reduce movement and prevent chafing. Adjust the valves so as to produce a pressure variation of about 15 cm of water. Clear away mucus by tilting, swabbing and using a sucker. The respirator may be opened for a few minutes for nursing purposes; the motor should not be stopped.

In case of difficulty the servicing agents are Messrs Stanley Cox Ltd. 11 Gerrard Street, Shaftesbury Avenue, London W 1. Telephone GERrard 5024.

OXYGEN THERAPY

Before describing the methods of administering oxygen a brief statement on the theoretical aspects of oxygen therapy is necessary because of the confusion which exists as to the indications for its use and what may be expected from it.

Oxygen lack or anoxia presenting as an emergency usually arises because blood is not properly oxygenated in the lungs. This is anoxic anoxia (often called anoxæmia). Since the hæmoglobin is not fully saturated with oxygen arterial blood is venous in character. 100 c cm blood contains 15 gm hæmoglobin which when fully saturated combines with 20 c cm oxygen. Normally

blood is 95 per cent saturated and so 100 c cm contains 19 c cm oxygen combined with hæmoglobin. In addition, 0.3 c cm is present in solution in 100 c cm plasma giving a total content of 19.3 c cm.

The causes of anoxic anoxia are all the conditions which interfere with pulmonary ventilation. Blood reaches all parts of the lung but air entry is defective and oxygenation incomplete. This type of anoxia is the best indication for oxygen therapy and is shown by cyanosis of recent origin. Anoxic anoxia may also develop at high altitudes because the partial pressure of oxygen in the inspired air is diminished. (*See Medical Emergencies in the Air page 347*.)

When we double the oxygen content of the inspired air (normally 20.96 per cent) we enable oxygen to diffuse through the alveoli at twice its usual rate and so enable the blood to be fully oxygenated through lungs which are ineffectively ventilated.

When only part of a lung is consolidated and the rest normal, oxygen will not improve the anoxia. This is because oxygen cannot reach the solid lung and the blood flowing through the healthy lung is already fully saturated with oxygen.

The other types of anoxia are **anæmic anoxia** (caused by a low total quantity of hæmoglobin albeit fully saturated with oxygen), **stagnant anoxia** (from slow blood flow), and **histiotoxic anoxia** (produced by poisons which interfere with respiratory enzymes). None of these are good indications for the use of oxygen though in high concentrations it may be helpful.

Size of cylinder and rate of flow.

The content of oxygen cylinders normally supplied is 20, 40, 100, and 150 cubic feet. A full oxygen cylinder gives a definite 'ring' when tapped but the only reliable method of finding out whether the cylinder is full is by using a pressure gauge. It is useful to know how long a cylinder will last. This can be worked out from the rate of flow. As this is expressed in litres per minute and the cylinder capacity in cubic feet it is necessary to remember that there are 28.3 litres to a cubic foot. Alternatively, if F = number of cu. ft. in the cylinder and M = the number of litres per minute of flow, then the cylinder will last

$$\frac{F}{28.3} \text{ hours}$$

Patients may be intolerant of any form of apparatus and a sedative such as paraldehyde which does not depress the respiratory centre may be needed before oxygen can be given

It has been found that at any given rate of flow of oxygen the alveolar concentration tends to vary inversely as the patient's vital capacity. This suggests that higher rates of flow are needed to maintain any given alveolar concentration in large patients than in smaller ones

METHODS OF GIVING OXYGEN

Nasal catheter or tubes.

A 7 to 12 Jacques catheter is used with several holes cut near the end. Measure the distance (usually about five inches) from the nares to the tragus and mark this on the catheter with ink, and insert the catheter to this mark. It should be lubricated with Nupercaine lubricant 10 per cent or similar anæsthetic ointment, and held in place by strapping or bandage.

A flow of three litres per minute is needed to double the alveolar oxygen (normally 14 per cent). Such a rate may cause irritation but this can be avoided by using two catheters connected by a "Y" piece. It is preferable to bubble oxygen through water in a Wolff's bottle or flow meter to counteract its drying effect on the throat. If a catheter is not tolerated, bicycle valve tubing may be used and held in place by a special spectacle frame. In another model, rubber tubing is dispensed with and replaced by malleable metal tubing which is shaped so as to lie about half an inch within the nares. Since in none of these methods is a reservoir provided between the nose and the oxygen cylinder, much oxygen is lost during expiration.

It is scarcely necessary to add that to administer oxygen by tube and funnel held near the face is futile though in a small infant a small rubber funnel held *closely* over the nose and mouth is useful as a temporary measure.

Facial mask.

Six models are generally available, all of which are efficient. Four are of the B L B type (Boothby, Lovelace and Bulbulian), and two are of the O E M type (Oxygen Equipment Manufacturing Co.), sometimes referred to as the Barach Eckman injector

mask. The essential differences between the B L B and O E M types are two —

1 In the B L B type oxygen is delivered through any type of flow meter into the bag whereas the O E M type utilises an injector type of mixing chamber to control the oxygen percentage

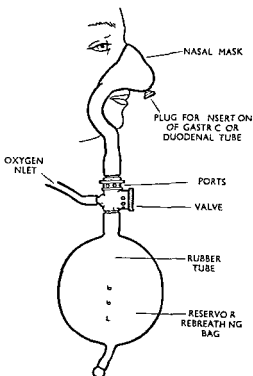


FIG 77

B L B mask Type 1 (nasal)

2 In the B L B type some re breathing into the bag occurs with a resulting higher CO_2 content of the air breathed than that of ordinary inspiration whereas with the O E M mask no re breathing takes place

B L B TYPES —Each has a moulded rubber face piece which must fit accurately. The malleable metal strips should be adjusted to ensure this. A rubber strap over the head holds the mask in position. The nasal type of mask leaves the mouth

free and is to be preferred but for patients with nasal obstruction an oronasal mask is available. Each model is made in two sizes and each of these may be obtained with a plug hole for a gastric or duodenal tube.

Type 1 (Fig. 77) —The patient breathes in from a bag connected to the oxygen supply and out through an expiratory valve. Atmospheric air can be admitted by opening one two

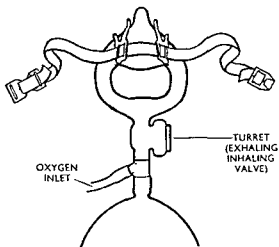


FIG. 78
B.L.B. mask Type 2 (nasal)

or three ports. With a flow of four litres per minute and all three ports open the alveolar oxygen concentration reaches 56 per cent. At six litres per minute and all ports closed it reaches 87 per cent.

Type 2 (Fig. 78) —This is similar to (1) but instead of a metal valve there is a disc of sponge rubber which acts in the same way.

Type 3 (Fig. 79) —Here the expiratory valve has been done away with and replaced by a seven mm. hole in the tube connecting the face piece to the bag. Oxygen enters the bag through a vent near the top and the patient breathes the gases from the bag mixed with air drawn in through the hole. On expiration air first passes into the bag and when this is distended it escapes through the hole.

At four litres per minute this model will raise the alveolar oxygen to 65 per cent the CO_2 in the bag rising to only 1.33 per cent

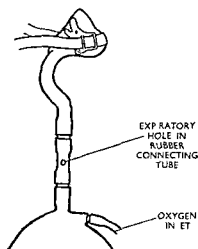


FIG 79
B L B mask Type 3 (nasal)

Fig 80 (Figs 80-81) —In this model the seven mm hole is on the face mask.

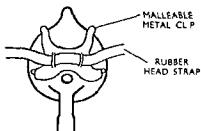


FIG 80
B I B mask Type 4 (oro nasal)

Further details of these masks can be obtained from L M S Memorandum No 5 (revised) 1944 Oxygen administration H M Stationery Office (Price 4d)

In the ordinary O.E.M. or Barach-Eckman mask (Fig 82) there is an expiratory valve on the face piece and a mica inspiratory valve at the entrance to the collecting bag. There is no re breathing and the bag is simply an oxygen reservoir. Nasal

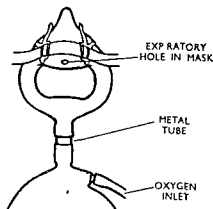


FIG 81
B L B mask Type 4 (nasal)

and oronasal types are made. Should the bag collapse completely there is a safety valve which opens to admit outside air until the bag inflates again.

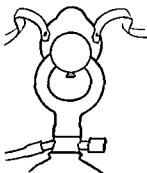


FIG 82
O E M mask (nasal)

The oxygen percentage is governed by an injector. This is a chamber (oxygen concentration meter, Fig 83) to which

oxygen is admitted through a restricted orifice. A negative pressure is caused in the rear of the chamber where there is a disc with different sized openings to admit air. The oxygen percentage of the mixture is stated at each opening. The CO content of the inspired air does not exceed 0.2 per cent. The tubing from the injector to the mask is $4\frac{1}{2}$ feet long and of $\frac{3}{8}$ inch internal diameter. These measurements must not be changed. A dial type flow gauge must be used with the injector and not a bobbin flow meter.

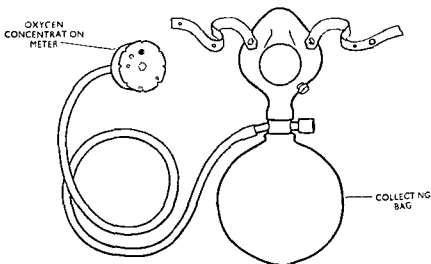


FIG. 83
O.E.M. mask (oxygen mask)

Another variety of O.E.M. mask (Fig. 84) is adapted so as to provide oxygen under positive pressure. This may be useful in pneumonia and cardiac failure but is not indicated when venous return to the heart is diminished as in shock. A disc with five graduated apertures, all smaller than the diameter of the larynx, is placed over the expiratory valve so that a positive pressure is reflected back into the lungs during expiration. With the largest orifice little or no positive pressure is produced. With the other orifices pressures of 1, 2, 3 and 4 cm. of water are

obtained when the disc is turned to the appropriate opening
 A new oro-nasal mask, the "Oxyair" made of plastic

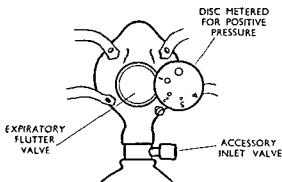


FIG 84

O E M mask (oro nasal with positive pressure attachment)



FIG 85

The Oxyair oro nasal mask made of plastic material

material (Fig 85), has been designed by Dr Basil S Kent, and is obtainable from Oxygenaire Ltd. An 11 mm orifice covered by phosphor bronze gauze acts as an inspiratory and expiratory valve. Its bag is made of rubber or polyvinyl chloride. Together

with its bag it weighs only $1\frac{1}{2}$ ozs. The plastic material is resistant to antiseptics but it cannot be boiled.

Improved mask

In an emergency an improvised mask may be made as suggested by Aitken and Cruikshank from a 15×12 inch X ray film from which the emulsion has been removed by hot sponging. The measurements are shown in Fig. 86.

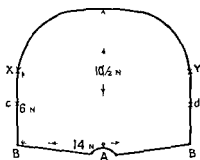


FIG. 86

Pattern for improvised mask

AB and AB are sewn together with about one inch overlap. The perforated rubber tube is attached along this junction for about four inches. XBBY is reinforced with copper wire and bound with lint, and the tapes are attached at X, Y, c and d. When the mask is applied to the patient the wire reinforced edge has to be moulded to give close contact with the skin under the chin and up to the level of the ears, and the mask should be adjusted to leave a space of about $2\frac{1}{2}$ inches between its upper part and the patient's forehead. All seams should be made air tight with strapping.

A simpler mask can be quickly made from a 15×12 inch film. Form a cone by bringing the corners of a long side together so that they overlap by about two inches. Stitch or clip the margins together and into the apex of the cone fix a rubber tube with strapping. Pad with gauze the edges that touch the patient's face.

Oxygen tents.

Tents are available for all types of patient including the new born (Figs 87 88 89) They are readily obtainable from



FIG 87

The Queen Charlotte infant tent (For use with premature and newly born babies)

Oxygenaire Ltd (Telephones —London WELbeck 1322 Bristol Abson 81 Birmingham Calthorpe 1737 and Manchester Sale 5620)

An experienced operator will bring the tent and set it up Soda lime to remove CO_2 is not necessary in most tents because CO_2 is lost by unavoidable leakage, especially at a high rate of

flow. If leakage is prevented and CO_2 absorbed much more supervision is necessary. The oxygen content inside the tent is



FIG 88

The Wigmore Junior tent

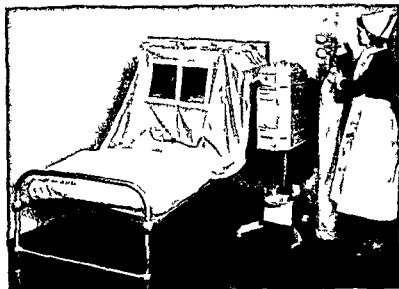


FIG 89

The Wigmore adult tent

easily measured by a simple analyser and if leakage is prevented should be tested every three or four hours

PRECAUTIONS—Oil and grease must not be used on taps and valves. Electrical apparatus of all kinds and anything liable to spark should be kept well away from the oxygen tent

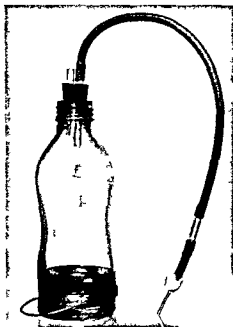


FIG 90
Blood collecting bottle

VENESECTION

No matter which method is used the preliminaries are the same. A tourniquet or sphygmomanometer cuff must be applied above the elbow tightly enough to obstruct venous return from the arm while not obstructing the radial pulse. A pressure of 80 mm Hg is sufficient. After cleansing the skin a few minims of 2 per cent procaine are injected over the vein selected at the bend of the elbow. Blood may be withdrawn—

- (1) By making a nick into the vein with a scalpel. The blood is received into a bowl. On completion raising the arm and a tight bandage over a pad stops the bleeding.
- (2) By a needle attached to a tube leading to a blood transfusion bottle (Fig 90). A French's needle (Fig 91) or a

Medical Research Council needle (Fig 92) is suitable. Some 3·8 per cent sodium citrate solution should be run through



FIG 91
French's venesection needle



FIG 92
Medical Research Council needle
(actual size)

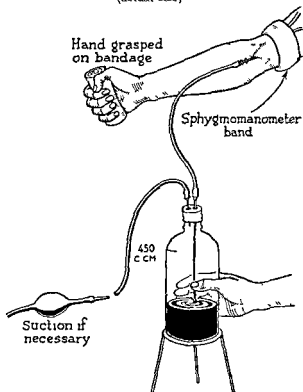


FIG 93
Showing the method of taking blood

the tube and needle before use to obviate clotting. If the bottle is of the type with a screw top and rubber disc blood

is admitted through a needle which pierces the disc (Fig 93) Suction may be applied to the bottle by a reversed Higginson's syringe attached to the air inlet

Blood taken in this way in a sterile bottle should be grouped and saved for future use, or for the preparation of plasma

A simple suction apparatus may be made from a bottle cork and two pieces of glass tubing as shown in Fig 94

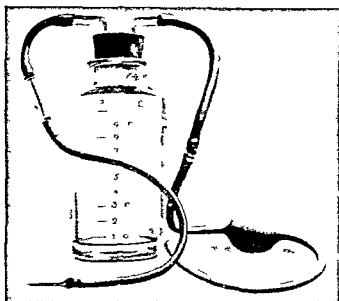


FIG 94
Venesection apparatus
(Pye Surgical Handicraft)

If the blood flow becomes feeble —

- (1) The tourniquet may need re adjustment
- (2) The angle at which the needle enters the vein may need changing
- (3) Suction may be required
- (4) Clamping and unclamping the hand may increase the flow
- (5) Clotting in the tubing may necessitate its removal

INTRAVENOUS INFUSION

Choice of the Vein

A vein in front of the elbow (Fig 95) is frequently used but

a leg vein is more convenient if the patient is restless (since splinting is easier than in the arm) or if veins are collapsed and

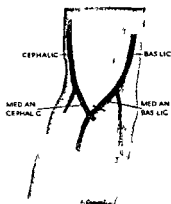


FIG 95

The median cephalic vein and
the median basilic vein
(*Surgery of Modern Warfare*)

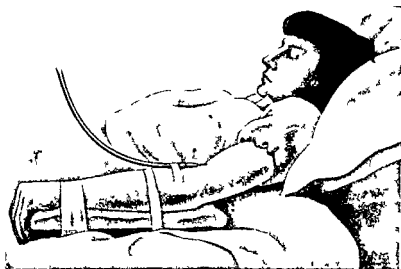


FIG 96

Arm fixed in pronation. This is more comfortable than the usual
supinated position. (*After J. I. Lecky*)

cutting down is necessary. If the arm is used it should be fixed to a splint in pronation which is more comfortable than the

supinated position (Fig 96) The most convenient leg vein is the anterior saphenous as it lies on the anterior surface of the tibia midway between the internal malleolus and the tendon of the tibialis anticus (Fig 97) This vein can often be seen and invariably palpated here A tourniquet above the knee may be needed to make it stand out



FIG 97

The long saphenous vein
(*Surgery of Modern Warfare*)

Needle technique

This is best reserved for the easily visible and palpable arm vein The tubing from the fluid container ends in an adaptor It is filled with fluid and clipped off The elbow is fully extended and well supported by a pillow and a venous tourniquet applied The vein is entered by the needle attached to a syringe so that by withdrawing some blood it is made certain that the needle is in the vein The needle point is pressed with a swab the syringe quickly detached the adaptor connected and the fluid allowed to flow

Cannula technique

EXPOSURE OF A VEIN—At the site of exposure—usually the ankle—2 per cent procaine is injected into the skin and subcutaneous tissues taking care not to damage the vein The skin is

lifted by toothed forceps (the vein being attached to the periosteum is left behind) and a transverse incision one inch long made over the vein

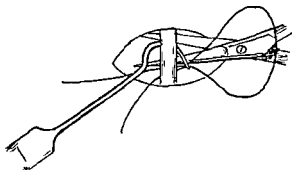


FIG. 98

Insertion of catgut under vein by aneurysm needle
(J H Kirtland)

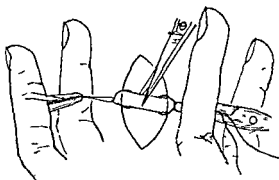


FIG. 99

Vein rendered taut by traction on catgut
(J H Kirtland)

About one inch of the vein is exposed by inserting closed sharp pointed scissors or a small hæmostat and opening the blades longitudinally. The vein is similarly freed from its periosteal attachments.

An aneurysm needle threaded with No. 0 catgut is inserted under the vein and the loop pulled through and cut so that two strands of catgut are left under the vein (Fig. 98). Each is tied

loosely by a single knot and both ends of each piece secured in Spencer Wells forceps

OPENING THE VEIN AND INSERTION OF THE CANNULA—The Spencer Wells forceps attached to the ligatures are held between the index and middle and the ring and little fingers respectively so that the vein is held taut (Fig 99). A transverse nick is then made

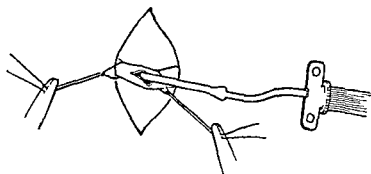


FIG 100
Insertion of cannula
(J H Kirkham)

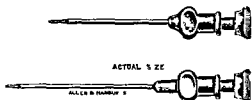


FIG 101
The Bateman needle

in the vein by scissors and the tension on the vein relaxed so that it can be seen if blood flows since sometimes the incision may not reach the lumen. A small longitudinal incision from the transverse one may also be made. The cannula (one of Hamilton Bailey's pattern of suitable size is convenient) (Fig 100) is then inserted with the fluid running slowly (Fig 100).

Another useful type is the Bateman double needle (Fig 101). For infants a ureteric catheter can be used. If the vein is small

it is an advantage to put a finger tip under it so that the end of the cannula can be pressed against it (Fig 102)

The fluid is then allowed to flow and when it is running satisfactorily the distal ligature is tied then the proximal one distally to the shoulder of the cannula and both are cut short. There should be as little delay as possible in letting fluid run from the container once the vein is entered since otherwise blood will enter the cannula and clot. The Bateman needle avoids this danger. When it is used it is best to connect it to the tubing by an adaptor.



FIG 102

Showing finger tip under vein and longitudinal and transverse incisions in vein wall

(J H Irlam)

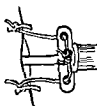


FIG 103

Showing method of tying in a Hamilton Bailey cannula

The skin incision is closed by a silkworm gut suture at each end. If a Hamilton Bailey cannula is used the distal end of each suture is passed through the holes in the wings of the cannula and tied (Fig 103). This ensures that the needle will not pull out of the vein. A gauze swab is placed between the cannula and the skin and another over the incision and secured by strapping.

The fluid container should be tied to the wall or a stand and need be only two feet or so above the level of the vein. A drip bulb must be placed in the tubing preferably nearer to the arm or leg than to the container.

SPECIAL POINTS TO BE OBSERVED IN THE CASE OF CHILDREN —

The foot and leg should be strapped to a splint on the outer side of the leg and extending from above the knee to 12 inches beyond the foot—i.e. it must be long enough completely to immobilise

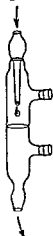


FIG 104
A drip bulb

the limb and the rubber tubing. The technique of exposing the vein is as already described.

Failure of flow.

If flow is unsatisfactory the following measures may set things right

- (1) See that the tubing is not kinked
- (2) See that the position of the cannula has not altered
- (3) Place hot water bottles on the limb (to diminish venous spasm)
- (4) Massage the limb upwards
- (5) Apply positive pressure to the inlet by a Higginson's syringe
- (6) If the above measures fail clotting in the cannula is likely detach the tubing or adaptor from it and try with a small syringe to clear it by suction

If the rate of flow cannot be observed because the drip bulb becomes full of fluid and the air column lost clip the tube above the bulb and detach it. Allow the fluid to run down in the bulb to a convenient level. Re attach the tubing and unclip it. A simpler method is to inject a little air into the rubber tubing near the bulb. In drip bulbs with a side arm air can be admitted through this by removing a cork (Fig 104)

SELECTION OF BLOOD FOR EMERGENCY TRANSFUSION

(1) By blood grouping using A and B sera

(a) Prepare a dilute emulsion of the donor's red blood cells by mixing two or three drops of blood obtained by a finger prick with 2.3 c.cms of normal saline or 3.8 per cent sodium citrate solution. Any other isotonic solution can be used and the degree of dilution does not matter greatly. The donor should be robust and should not have suffered recently from any infective fever, jaundice or malaria or from syphilis.

(b) Place two drops each of A and B serum on a glass slide or white tile and mark them A and B respectively with a grease pencil.

A serum is obtained from Group A(II) blood and contains the agglutinating substance or agglutinin β . Agglutination is thus seen when it is mixed with cells of Group B blood (containing the agglutinable substance or agglutininogen B). Hence A serum is sometimes labelled Anti B serum. Similarly B serum is obtained from Group B blood and contains α agglutinin. It reacts with A cells and may be labelled Anti A.

(c) Add two drops of the dilute emulsion of cells to each drop of serum and mix thoroughly. A match or glass rod can be used but care must be taken to use a separate one for each drop.

Rock the slide gently. Agglutination is visible to the naked eye and usually appears within five minutes but may take up to half an hour. The group is indicated by the result as shown in Fig. 105.

If no agglutination occurs with either serum it is wise to wait another ten minutes and to look for agglutination under the microscope before saying that the donor belongs to Group O (IV).

(2) By direct matching

Except in cases of extreme emergency it is wise to test the recipient's serum against the donor's cells as well as to determine their groups.

Two to 5 c.cms of the recipient's blood are taken and the serum obtained by allowing it to clot or by centrifugalisation. This serum is mixed with a dilute suspension of donor's cells as described above. Agglutination indicates probable

incompatibility Transfusion should not then be undertaken unless an experienced pathologist has proved the agglutina

STOCK SERUM A STOCK SERUM B BLOOD BELONGS TO



Agglutination



Agglutination

GROUP I - A B



No Agglutination



Agglutination

GROUP II - A



Agglutination



No Agglutination

GROUP III B



No Agglutination



No Agglutination

GROUP IV O

FIG 105

Blood grouping The result obtained in each of the four groups is shown (colour photograph)
(*Surgey of Modern Warfare*)

tion to be due to factors other than true incompatibility
(e.g. cold agglutinins)

Except in emergencies it is better for all blood grouping and cross matching to be undertaken by a pathologist experienced in the technique and familiar with the many difficulties that may arise



FIG. 106

In order to mix the constituents it is necessary to invert the bottle to see only

Scenes of Modern Warfare

The Rh factor

If a patient is Rh negative pregnancy or transfusion of Rh positive blood may stimulate the production of Rh antibodies. Subsequent transfusions may be followed by hæmolytic reactions. If a patient therefore has been pregnant has had previous transfusions or is likely to need further transfusions it is wise to determine the Rh group in the laboratory. If she is Rh negative only Rh negative blood should be given.

BLOOD TRANSFUSION

Two methods are available the direct or arm to arm method and the indirect. The latter is most commonly used. Blood is taken into a bottle as described under Venectomy (p. 421) and used fresh or after storage.

The technique of entering the vein is exactly as described

under intravenous infusion. Special points in the transfusion of blood as opposed to other fluids are —

- (1) The bottle must not be shaken but merely inverted once or twice



FIG 107

The cap is unscrewed and the beads are poured into the bottle from the sterile test tube

(*Surgery of Modern Warfare*)

- (2) If the bottle is taken straight from a refrigerator it should be placed in water at 99°F but not heated more than this lest clotting occurs
- (3) Glass beads or a gas mantle filter must be inserted into the bottle before use

Stored blood should be rejected if there are signs of hæmolytic *i.e.* a definite red tinge in the supernatant plasma. It should also be rejected if the plasma is turbid as this indicates infection. The various steps in technique are illustrated in Figs 106 to 111 taken from '*Surgery of Modern Warfare*' by kind permission of Mr Hamilton Bailey



FIG 108

The perforated cork with its attached tubing is inserted into the bottle firmly. The bottle is then inverted and it is
(*Surgery of Modern Warfare*)



FIG 109

handed to the Sister who hangs it on a stand. She then applies a pair of Spencer Wells forceps to the tubing above the interceptor and releases the screw clamp completely.
(*Surgery of Modern Warfare*)

Blood transfusion by the direct (arm to arm) technique

All that is required is a Jube syringe (Fig 112) some 3.8 per cent sodium citrate solution a tourniquet a sphygmomanometer some 2 per cent procaine and a hypodermic syringe and instruments for cutting down on a vein.

The arms are inspected and the patient and donor brought



FIG. 110

The operator having scrubbed up holds the needle or cannula well above the level of the table and he prepares to catch the blood issuing from the needle with a syringe in his free hand. The Sister has released the part of Spencer Wells. Blood is issuing from the cannula and if held like this air in the tubing is quickly expelled. The Sister then reapplies the Spencer Wells forceps to the tubing.

By J. of M. I. H. a /

alongside on tables so that the donor's better arm is next to the patient. The arms are supported on a small table or high stool.

A tourniquet is applied to the recipient's arm and the sphygmomanometer cuff to the donor's arm. The skin is cleansed and towels clipped over the hands and upper arms.

The special Jube trocar and cannula is inserted into the donor's vein first (after a wheal of 2 per cent procaine has been

raised and a small scalpel nick made) The trocar is removed to show that blood flows and quickly replaced and the cannula fixed by strapping

The recipient's vein is similarly dealt with or exposed first by incision if necessary The syringe and tubing filled with 3·8 per cent sodium citrate is taken from its bowl The donor's



FIG. 111

The cannula having been inserted into the vein the self-releasing tourniquet having been dropped and the Spence Wells forceps removed from the tubing the blood is flowing rapidly into the vein It is necessary to adjust the number of drops which are passing through the interceptor and the whole procedure is completed

(Courtesy of Mr. L. J. Warfar)

trocar is removed and the adaptor attached to the tubing quickly fitted to the cannula The syringe is then likewise connected to the recipient's cannula The recipient's tourniquet is removed

With the syringe's piston slot to the donor's side (*see page 41*) the barrel is quickly filled the piston is turned so that the slot faces the exit tube and the blood is injected quickly into the recipient The procedure is repeated until the desired amount of blood has been given The barrel holds 5 c.c.m. and a nurse should be instructed to count the piston strokes

Bone marrow infusion in infants.

The most convenient site is the subcutaneous surface of the tibia at least an inch below the tibial tuberosity so as to avoid the epiphyseal line. The leg is externally rotated so that the flat subcutaneous surface of the bone looks directly upwards. It

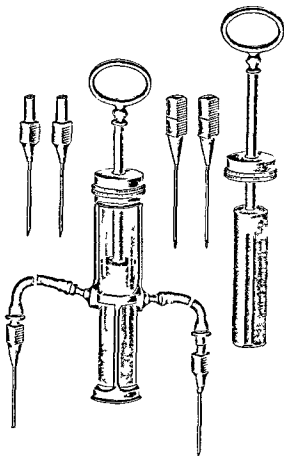


FIG. 112
The Jubé syringe

is splinted in this position. A convenient way is to tie both feet nine inches apart to a splint which is fixed at each end to the bed. Movement of the limb is restricted by sandbags.

A full aseptic technique must be adopted. After cleansing the skin the tissues down to the periosteum are infiltrated with

2 per cent procaine. Several types and sizes of needle are available and that of Behr (Fig. 113) is recommended.

It is inserted at right angles to the bony surface with a boring movement and a definite crunch is felt when it penetrates the cortex. After withdrawing the stylet some marrow

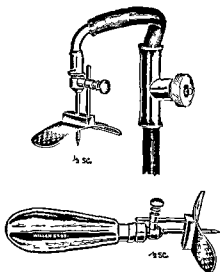


FIG. 113
Behr's tibial trocar set

may be sucked up with a syringe. It is a wise precaution to inject a little saline with a syringe to make sure that the needle point is in the marrow and not in the soft tissues. The tubing filled with transfusion fluid is attached to the needle by an adaptor and the fluid allowed to flow. By means of the flange the needle is tied firmly to the limb.

In a new born baby a rate of flow of half an ounce an hour is about the maximum but faster rates can be attained in older children. If the rate is too slow the fluid container may be raised six to eight feet above the leg. Should the needle become loose and fluid leak back into the subcutaneous tissues the infusion must be stopped.

The infusion can be kept going for 48 hours or longer. The needle should not be removed too soon since a second transfusion

cannot be given again into the same bone for some days because fluid would leak back through the original hole

ANTI-COAGULANT THERAPY

When it is desired to diminish the coagulability of the blood as, for example, in thrombo phlebitis or coronary or cerebral thrombosis and embolism two drugs are available—Heparin and Dicoumarin

Heparin causes an immediate lengthening of coagulation time which quickly falls to normal when the drug is stopped. It must be given intravenously. Dicoumarin acts only after a latent period of 24 to 48 hours and its effects continue for about the same time after it is discontinued. It is active when taken by mouth. Heparin but not dicoumarin is active *in vitro*.

HEPARIN

This is best given as the barium salt by intravenous drip in doses of 100 to 300 mgm daily. The dose should be regulated by estimating the coagulation time. Normally $1\frac{1}{2}$ to $1\frac{3}{4}$ minutes it should be raised to between 10 and 15 minutes. The effects of heparin can be reversed quickly if necessary by injection of protamine sulphate (salmine) in doses of 5 mgm per kilo body weight. (It is not generally available but small amounts can be supplied by Burroughs Wellcome & Co and by Eli Lilly & Co.)

DICOUMARIN

The prothrombin time of undiluted plasma is first estimated—a laboratory procedure. It is normally 13 to 17 seconds. Dicoumarin 300 mgm (dispensed in 50 mgm tablets) is then given by mouth and repeated daily until the prothrombin time is about doubled. This necessitates a daily prothrombin estimation before the dose is administered. The prothrombin time should be kept at about 30 seconds by daily doses of 100 to 200 mgm. If necessary the drug can be given as the disodium salt intravenously.

Overdosage causes severe bleeding from many places. Should this occur it can be countered by blood transfusion, preferably using fresh blood by the arm to arm method (*page 435*). Another remedy is a large dose (60 to 250 mgm) of a synthetic

vitamin K analogue called Synkavit (Roche) which being water soluble can be given intravenously. Contra indications to the use of dicoumarin are hepatic and blood diseases.

SULPHONAMIDE THERAPY

Sulphonamides should be given in the doses shown in the table (Fig 114). If the patient cannot swallow the tablets (usually 0.5 gm) they may be crushed and administered through a stomach tube. In the case of a child whose weight is known the dose can be calculated on the basis of 1 grain per lb per 24 hours. Broadly speaking the maximum safe daily dose for an adult is 10 to 12 gm and for the smallest child 3 gm. Most sulphonamides have to be given four hourly but sulphamerazine is maintained at an adequate blood level by eight hourly administration because of its slow excretion.

Parenteral administration

For this purpose the sodium salts of sulphonamides are used (There is no sodium salt of sulphanilamide). As these salts are strongly alkaline and therefore irritant they should not be given intramuscularly or intrathecally but only into the blood stream where rapid dilution can occur.

Soluthiazole (May & Baker) a special readily soluble compound does not have these drawbacks as its solution has a pH of 7. It is supplied as a 45 per cent solution (5 c cm containing the equivalent of 1 gm of sulphathiazole).

Soluble sulphonamides must not be added to blood or plasma transfusion apparatus because they cause precipitation of protein and blockage of tubing.

Fluid intake

An adequate urinary output [i.e. at least 50 fl oz (1500 c cm) in 24 hours in an adult] must be maintained during sulphonamide therapy though this is not so important in the case of sulphanilamide and sulphacetamide which do not cause urinary obstruction. A fluid intake of about six pints in 24 hours is necessary in an adult. The urine should be kept alkaline to litmus by giving 20 gr each of sodium bicarbonate and sodium citrate with each dose of sulphonamide. The fluid requirements of a child may be worked out on the basis of one pint for every gram of sulphonamide given.

“ Sulpha combination ”

Each sulphonamide and its acetyl derivative crystallises out independently in the urinary tract. It has been shown that a mixture of sulphonamides may be safely given without risk of deposition in the kidneys in doses double those of any one sulphonamide which could ordinarily be used. The mixture recommended in this sulpha combination consists of sulpha thiazole 37 per cent sulphadiazine 37 per cent and sulpha merazine 26 per cent (Sulphatriad tablets 0.5 gm. May and Baker).

The poorly absorbed sulphonamides—succinyl sulphathiazole phthalyl sulphathiazole and sulphaguanidine—used for intestinal infections are given in doses of at least 12 gm. daily but this dose may be greatly exceeded without harm.

PENICILLIN THERAPY

The emergencies for which penicillin is indicated are described in the text. Here some practical points in its administration are considered.

Although it is inadvisable when penicillin seems indicated in an emergency to delay its use until the causal organism is identified steps should be taken to provide this evidence by taking blood cerebro spinal fluid etc. before the first injection is given.

Preparations

What we regard as pure penicillin is a mixture of several similar compounds of varying antibiotic activity. It is a white crystalline substance and has a potency of 1 650 units per mgm. An international unit is the activity contained in 0.6 microgram of pure penicillin.

Penicillin is supplied as the dry (yellow) sodium salt and as white crystalline penicillin in rubber capped vials containing 100 000 200 000 and 500 000 units and 1 000 000 units (— 1 mega unit). Sterile water or normal saline is injected into the vial in such an amount that the dose to be given will be contained in from 1 to 2 c.c.m. It is preferable to remove all traces of alcohol or other antiseptic from the syringe but if all the contents of the vial are given at one injection a trace of antiseptic in it does not matter. Penicillin is very soluble and a massive dose of

500,000 units can be administered in 2 c cm of fluid. Aqueous solutions remain active for 14 days at room temperature under sterile conditions and longer in a refrigerator. Penicillin lozenges contain 500 units each.

Dosage.

Since penicillin must usually be given by injection infrequent doses are preferable to the patient but whereas an effective blood level can be maintained by 25 000 units three-hourly the duration of effect cannot be doubled by simply doubling the dose. A dose of 200,000 units night and morning is commonly employed but even this dosage leaves the patient with an insufficient amount of penicillin in his blood for a large part of the interval between doses.

In recent years attempts have been made to produce a more sustained action by incorporating the penicillin in a base of bee wax and arachis oil (300,000 units per c cm). In practice however, this vehicle for penicillin is awkward to use; the container must be immersed in hot water to allow the wax medium to melt, and the syringe must be heated. The harmful effects of waxes and oils on the tissues have also been pointed out recently.

Should injection be found to be painful 0.5 to 1.0 c cm of 2 per cent procaine should be taken up into the syringe *after* the penicillin so that it precedes the latter into the tissues when injected.

Being for all practical purposes non-toxic there is no disadvantage, other than expense, in giving a big dose. Dosage for children need not differ greatly from that given to adults. It should not be less than 4 000 units per lb of expected body weight per 24 hours.

Route of administration.

At the time of writing, the relative advantage of a continuous intramuscular penicillin drip, three-hourly intramuscular injections of small doses, or larger doses at longer intervals are unsettled. In an emergency, however, there is nothing to be lost by giving a large dose intramuscularly in the first place.

Intrathecal penicillin

Penicillin does not reach the C S F from the blood stream

in effective concentration and so if used in meningitis it must be given intrathecally. Pure crystalline penicillin must be used. Daily or twice daily injections of 8 000 to 16 000 units should be given using a solution containing 2 000 units per c cm. More concentrated solutions may be harmful.

Penicillin by mouth

Penicillin is absorbed from the duodenum and reaches the blood in effective levels provided the dose is 5-10 times the intramuscular dose and that it is given before breakfast or four hours after a meal. The best vehicle is 10 per cent glucose. For bottle fed infants penicillin should be added to the first half fluid ounce of the feed.

Penicillin by inhalation

A suitable solution for atomisation and inhalation is one containing 100 000 units per c cm. One or two c cm should be inhaled during a period of 15 minutes every three or four hours. Since yellow penicillin has a mouldy odour the crystalline product is preferable. For children inhalation is best achieved by connecting a Collison inhaler to an oxygen tent. A separate cylinder must supply oxygen in the usual way.

Atomising machines are of two types —

- 1 Those operated by compressed air or oxygen such as the Oxylin inhaler (Oxygenaire Ltd 8 Duke Street W 1 Telephone WELbeck 1322) and the Collison inhaler (Inhalation Institute 87 Eccleston Square S W 1 Telephone VICToria 1676 Telegrams Idac London)
- 2 Those operated by an electrically driven diaphragm pump such as the Phantomyser M A 3 Model (Aerosols Ltd 65 Old Brompton Road S W 7 Telephone KENington 7495)

Failing these machines a hand atomiser may be used but as the particles are relatively large much of the material does not pass beyond the upper respiratory passages.

Penicillin reactions

These are mostly urticarial and resemble those following the use of serum substances. They may be immediate or delayed for three weeks. Treatment should be by adrenaline 0.5 c cm subcutaneously followed by ephedrine gr $\frac{1}{4}$ to $\frac{1}{2}$ by mouth and

Antistin (Ciba) 1 to 2 c cm intramuscularly three times a day together with the application of calamine lotion

STREPTOMYCIN

Streptomycin acts on many gram negative organisms which are mostly penicillin resistant such as *B coli typhoid* and dysentery organisms *haemophilus influenzae* and the tubercle bacillus

It may be administered by all routes including the mouth. Intramuscular injection is preferable and is followed by wide distribution throughout the body though as with penicillin concentration in the C S F is low. Intravenous injection may cause circulatory collapse and thrombophlebitis and is not recommended

Streptomycin is supplied in phials as the hydrochloride which is a white powder. It is readily soluble in water and normal saline. Since it is not a pure product the weight of powder does not correspond to the weight of pure streptomycin stated on the label. Hence the contents of the phial should be dissolved and an aliquot part of the solution used according to the desired dose. Streptomycin powder may be stored in the dry form without refrigeration for a year. Solutions are relatively thermostable but should be kept in a refrigerator and used within a week of preparation

Dosage

This depends on the sensitivity of the organism and it is recommended that a blood concentration of streptomycin should be maintained of four to eight times that necessary to inhibit the organism *in vitro*. The safe maximum daily dose by intramuscular injection is from four to six grams given in a concentration of 100 to 175 mgm per c cm. For intra thecal injection up to 100 mgm in 5 to 10 c cm of normal saline every 24 hours is adequate

A complete list of hospitals in England and Wales at which streptomycin treatment of tuberculous meningitis, military tuberculosis and influenzal meningitis is available can be obtained from the Ministry of Health Whitehall London S W 1 (Telephone WHItchall 4300). The local Medical Officer of Health could probably give the name of the nearest hospital

In Scotland The Department of Health St Andrews House Edinburgh 1 (Telephone Edinburgh 33433) and in Northern Ireland The Chief Medical Officer Ministry of Health and Local Government Stormont Belfast (Telephone Belfast 63210) should be approached

If streptomycin is wanted for non tuberculous cases other than influenzal meningitis enquiries should be addressed to Professor Clifford Wilson The London Hospital London E 1 (Telephone Bishopsgate 8333)

EMBALMING

In the case of a person dying suddenly when far from home or aboard ship the doctor may have to embalm the body quickly especially in a hot climate It is as well to wait for 24 hours until rigor mortis has passed off Two gallons of the following solution should then be injected upwards into the femoral artery —

Formalin (40 per cent)	4 pints
Carbolic acid (crystalline)	1 lb
Water	2 gallons

About half a pint of this fluid should be injected into the pleural and peritoneal cavities The body should then be washed with a saturated solution of mercuric chloride in methylated spirit mixed with five times the amount of glycerin

If an autopsy is requested it should not be carried out for 24 hours after the embalming

If death occurs at sea and preservation of the body is requested it should be kept in a cold place with blocks of ice and not in the hold which is often warm Stench may be mitigated as described on *page 335*

C ALLAN BIRCH

Appendix I

SUPPLIERS OF ANTI VENINES FOR SNAKE BITE

Great Britain

Allen & Hanburys Ltd 7 Vere Street London W 1 Telephone MAYfair 2216

(Anti venine against the common adder *Vipera berus* the only poisonous snake in Britain)

South Africa

The South African Institute of Medical Research

(i) P O Box 1038 Johannesburg Telephone 44 1444 Telegrams Bacteria

(ii) Buckingham Road Port Elizabeth Cape Telegrams Bacteria

(iii) 7 Roth Avenue Bloemfontein Orange Free State Telegrams Bacteria

The Institute also supplies serum to the Government Departments of Nigeria Gold Coast Belgian Congo French Equatorial Africa Portugese East and West Africa French Cameroons and the numerous missionary stations in Central Africa

(Polyvalent anti venine against crotaline and lachesine vipers)

South America

Laboratorios Butantos 34 Rua Gloria Sao Paulo Brazil Telephone Sao Paulo 2 1788

(Polyvalent anti venines against crotaline and lachesine vipers)

United States of America

Mulford Laboratories 38 and Ludlow Street Philadelphia Pa Telephone Evergreen 1244

(Anti venine against North American pit vipers and Central and South American lachesine vipers Supplies very small because of limited demand)

India

The Central Research Institute Kasauli Punjab Telephone 11 Telegrams Problem

(Polyvalent anti venine against the cobra and Russell s viper)

The Director Haffkine Institute Parel Bombay 12 Telephone 60 084 Telegrams Research

(Lyophilised polyvalent anti snake venom serum against Cobra Common Krait Russell s Viper and the Saw scaled Viper)

Australia

Commonwealth Serum Laboratories Parkville N 2 Victoria Telephone FW 2101 Telegrams Serums Melbourne

Australian Capital Territory

Director General of Health Canberra Telephone Canberra 596 Telegrams Health Canberra

New South Wales

Senior Commonwealth Medical Officer Customs House Circular Quay Sydney Telephone B 6557 B 6558
 The Medical Officer in-Charge Health Laboratory Lismore Telephone Lismore 346 P O Box No 332

Victoria

Medical Officer in Charge Health Laboratory Bendigo Telephone Bendigo 867

Queensland

Senior Commonwealth Medical Officer Anzac Square Adelaide Street Brisbane Telephone B 1341
 Medical Officer in Charge Health Laboratory Townsville Telephone Townsville 242
 Medical Officer in-Charge Health Laboratory Toowoomba Telephone Toowoomba 914
 Medical Officer in-Charge Health Laboratory Rockhampton Telephone Rockhampton 3404
 Medical Officer in Charge Health Laboratory Cairns Telephone Cairns 2467

South Australia

Senior Commonwealth Medical Officer C M L Buildings 41 47 King William Street Adelaide Telephone Central 4898
 Medical Officer in Charge Health Laboratory Port Pirie Telephone Port Pirie 353

Western Australia

Senior Commonwealth Medical Officer 4th Floor G P O Perth Telephone B 6484
 Medical Officer in-Charge Health Laboratory Box 200 P O Kalgoorlie Telephone 173

Tasmania

Senior Commonwealth Medical Officer Health Laboratory Launceston Telephone Launceston 1072
 Medical Officer in Charge Health Laboratory 24 Campbell Street Hobart Telephone Hobart 6285
 (Tiger snake anti venines)

Appendix II

DRUGS MENTIONED IN THE TEXT AND THEIR EQUIVALENT PREPARATIONS

Adrenaline (Solution of Adrenaline hydrochloride B P 1 in 1 000

Adrenaline in oil (Parke Davis & Co) 1 c cm 2 mgm

Adrenaline hydrochloride (1 m 30 Solution of adrenaline hydrochloride B P)

Hyperdure Ad (Allen & Hanbury) Adrenaline with mucic acid Dose 3 to 8 m (0.18 to 0.5 c cm)

Adrenutol (Evans) There are two solutions —

(a) Adrenaline hydrochloride 1 in 500 and

(b) Adrenaline hydrochloride 1 in 1 000 both made up with chlorbutol 1 in 500 in water and glycerin (dose 1 c cm)

Kadamylin (Zimmerman) Adrenaline with pituitary extract (Formerly called Asthmolysin)

Adrenal cortex preparations

GLANDULAR EXTRACTS

Extract of suprarenal cortex B P C

Eucorfone (Allen & Hanbury)

Cortin (Organon)

Eschatin (Parke Davis)

Supracort (Paines & Byrne)

SYNTHETIC (Desoxycorticosterone acetate)

Cortenil (Bayer)

Cortigen (Richter)

Cortiron (British Schering)

D O C A (Organon)

Percorten (Ciba)

Syncortyl (Roussel)

Amethocaine hydrochloride B P. (Butethanol).

Anethaine (Glaxo)

Decicaine (Bayer)

Pontocaine (Winthrop, New York)

Tetracaine hydrochloride U S P XII

Carbachol B P.

Doryl (Merck)

Moryl (Savory & Moore)

Choryl (Pharmaceutical Products)

Dicoumarin (called Dicoumarol in U S A)

Temparin (Herts Pharmaceuticals)

Dicoumarin (Ward Blenkinsop)

Diodone

Perabrodil (Bayer)

Pyclosil (Glaxo)

Ergotamine tartrate

Femergin (Sandoz)

Neo femergin (Sandoz)

Hexobarbitone soluble B P

Hexanastab (Boots)

Cyclonal sodium (May & Baker)

Iodised Oil B P.

Iodatol (B D H)

Iodinal (Martindale)

Iodipin (Martindale Merck)

Lipiodol (Bengue)

Neo hydriol (May & Baker)

Iodoxyl B P

Uroselectan B (British Schering)

Pyelectan (Glaxo)

Pylumbrian (Boots)

Uropac (May & Baker)

Leptazol B P.

Cardiazol (Knoll)

Phrenazol (Boots)

Nikethamide B.P.

- Coramine (Ciba)
- Anacardone (B D H)
- Corvotone (Boots)
- Nicamide (Burroughs Wellcome)

Oestrogenic hormones. (Only solutions for intramuscular use)

- Oestroform (British Drug Houses)
- Dimenformon (Organon)
- Oestradiol benzoate (Burroughs Wellcome)
- Ovocyclin P (Ciba)
- Progynon B Oleosum Forte (British Schering)
- Theelin (Parke Davis & Co)
- Unden (Bayer)

SYNTHETIC PREPARATIONS

- Hexoestrol
 - Stilboestrol dipropionate
- Also supplied in tablets for
oral use

TABLETS FOR ORAL USE

- Dienoestrol
- Ovocyclin (Ciba)

Papaveretum B.P.C. Dose gr $\frac{1}{2}$ to $\frac{1}{4}$

(Not to be confused with **Papaverine B.P.C.**, dose two to four grains, which has mild hypnotic but marked antispasmodic effects)

- Omnopon (Roche)
- Alopon (Allen & Hanbury)
- Opoiloid (Richter)
- Opoidine (Macfarlane)
- Pavopin (T & H Smith)

Pentose nucleotide.

- Pentide (Allen & Hanbury)
- Pentnucleotide (Menley & James)
- S P N (Evans Medical Supplies Ltd)

Pethidine (called Demerol in U.S.A.).

- Dolantin

Procaine hydrochloride B P

- Novocain (Bayer)
- Novocaine (Saccharin Corpn)
- Parsetic (Parke Davis & Co)
- Planocaine (May & Baker)
- Sevicaine (Glaxo)

Progesterone Oily solutions for intramuscular injections
(containing 1 to 10 mgm per ampoule)

- Proluton (British Schering)
- Progestin (Organon)
- Progestin (British Drug Houses)
- Progesterone (Burroughs Wellcome)
- Lutocyclin (Ciba)
- Luteostab (Boots)
- Lutren (Bayer)
- Lipo Lutin (Parke Davis & Co)
- Gestone (Paines & Byrne)
- TABLETS 5 to 10 mgm) FOR ORAL USE
- Proluton C (British Schering)
- Progestoral (Organon)
- Ethisterone (B D H Boots)
- Gestone (Oral) (Paines & Byrne)

Pyridoxin (Adermin vitamin B 6)

- Benadon (Roche) 20 mgm tablets and 50 mgm ampoules

Theophylline and Ethylene diamine B P (— Aminophylline)

- Cardophyllin (Whiffen) (Formerly called Euphyllin)

Thiopentone soluble B P

- Pentothal sodium (Abbott)
- Evipan sodium (Bayer)
- Intraval sodium (May & Baker)

Vitamin C (Ascorbic acid B P)

- Cantan (Bayer)
- Celn (Glaxo)
- Davitamon C (Organon)
- Redoxon (Roche)

Vitamin B (Aneurine hydrochloride B P)

Befortiss (Vitamins Ltd)
 Benerva (Roche)
 Berin (Glaxo)
 Betahn (Lilley)
 Betaxan (Bayer)
 Davitamon B (Organon)

Vitamin E (Tocopheryl acetate B P C)

Davitamon E (Organon)
 Ephynal (Roche)
 Fertitol (Vitamins Ltd)
 Phytoferol (British Drug Houses)
 Viteohn capsules (Glaxo)
 Zygon (Squibb)
 Trigol (Abbott)

Vitamin K**NATURAL**

Klotogen (Abbott)

SYNTHETIC ANALOGUES**Oil soluble**

Acetomenaphthone B P

[Kapilon (Glaxo)]

Menaphthone B P

[Kappaxan (Bayer)]

[Prokayvit (B D H)]

Water soluble

Menadione bisulphate (U S P \II)

Synkavit (Roche)

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